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Section for the Study of Disease in Children.

President—J. D. Rolleston, M.D.

Provincial Meeting at the Royal Alexandra Hospital for Sick Children, Brighton, July 2, 1927.

The Treatment of Ringworm by Thallium Acetate.

By J. H. Twiston Davies, M.B.

The secret of success with thallium lies in the after-treatment, and for this reason one gets better results in private work than with hospital out-patients. In every case the infected hairs have to be extracted by hand, and this must be done on the day on which the hair first becomes sufficiently loose. The regrowth of new hair is occasionally so rapid that a delay of two or three days may endanger the result.

My practice is to give 8·75 mgm. thallium acetate per kilo and instruct the mother to rub Whitfield's ointment into the unaffected parts of the scalp only, and to wash the head every other day for the first week and daily for the second. The child should be brought up again when the hair is sufficiently loose to be pulled out in tufts. The hair is not loosened simultaneously all over the scalp, but when a clearing can be made easily round any infected patch, that is the time to extract the infected stumps. I prefer to do this carefully with lens and forceps where time is no object, otherwise the use of strapping is fairly efficient. As epilation progresses the bald areas are painted three times daily with 4 per cent. iodine in rectified spirit for four days and the whole scalp rubbed with sulphur ointment for the rest of the week, this sequence being repeated for about four weeks.

As regards the thallium, I have experienced only the slightest toxic effects in its application to normal children with tinea, consisting of pains in the joints of the lower extremities between the fifth and tenth days.

In the case of one child, who, as the result of an arithmetical error, was given a double dose (child weighing 3 st. given 5 gr.) the toxic effects were equally slight and did not appear till the tenth day, nor did epilation take place earlier than usual.

On the contrary—and this is the point to which I want to draw attention—patients with alopecia areata are hypersensitive to thallium. I was tempted by the experimental results of subepilating doses and by my own observation of the increased rate of growth of the hair in the period preceding therapeutic defluvium to give subepilating doses in three cases of alopecia areata. In the first case, that of a boy, aged 13 years, with alopecia areata universalis of twelve months' standing, I gave 6 mgm. per kilo. He appeared at my out-patient department a month later with the story of having been unable to stand and having been confined to bed with a cradle over his legs on account of severe pain in his knees and ankles. I excluded, as far as possible, acute rheumatism. The only improvement as regards the alopecia areata was the appearance of a few eyebrows.

In another case, that of a girl, aged 8 years, in whom the alopecia areata had preceded and persisted after cure of tinea by X-rays, the patient received 4 mgm. per kilo. The alopecia became total within a fortnight (sparing the "fringe") and has remained so for nearly three months, a little lanugo, however, appearing only on the parts not previously affected by the alopecia areata, on which also the follicles retained their normal appearance. She had some pain in the legs, but not sufficient to keep her in bed.

A Case of Fits.

By Gladys Wauchope, M.D., M.R.C.P.

R. G., aged 1 year and 9 months. Minor epilepsy associated with unsteady gait, deafness, ? mental defect. Born September, 1925, at term, weight at birth 6 lb. Cut first tooth at 6 months; stood at 11 months, walked at 12 months. Parents healthy, mother aged 42 when patient was born. Two sisters, aged 13 and 11 years, the elder has had rheumatic

fever with pericarditis and chorea and is nervous. No family history of epilepsy, asthma, urticaria, etc. No infantile convulsions in patient or sisters. Patient breast-fed for two months; afterwards on glaxo.

December, 1925.—Aged 3 months. Eczema developed on forehead and arm, and persisted in spite of treatment till April, 1926, when patient was taken to Switzerland and put on a diet of oatmeal and water.

July 21, 1926.—First seen for "attacks." A fortnight previously he had twitched his face and hands, rolled his eyes and had become drowsy. Twelve days later, onset of present type of attack. He stops his play, stares and falls or "droops" forward, recovering at once. This had occurred two or three times at about half-hour intervals, morning and evening each day after waking from sleep. On examination the child was bright-looking, thin (weight 19 lb.) and showed no signs of rickets or other organic disease. The diet of oatmeal and water with one egg a day was changed to almata (on account of the supposed idiosyncracy to milk), eggs, raw meat juice, bread and butter, etc. Bromide (1 or 1½ gr. three times a day) was given.

September, 1926.—Attacks slighter and less frequent. Gain in weight 2½ lb.

October, 1926.—Attacks more frequent in spite of bromide, 7 gr. per day. Seen by Dr. Macnamara, who noted diminution of consciousness and advised alternating luminal (⅛ gr. *ter in die*) with bromide.

December, 1926.—Noticed to be unsteady in his gait.

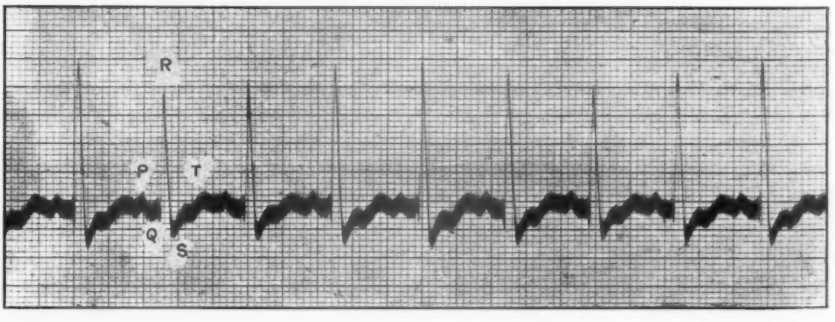
May, 1927.—Seen after five months interval: appeared to be mentally defective. Seen by Mr. Crow for suspected deafness. Hearing found deficient but not absent. High tones better appreciated than low.

Present Condition.—Attacks less frequent since June 6, when administration of bromide was resumed (2 grs. t.d.s.). Now one to three a day, occasionally none, generally after waking in the morning. Sometimes clenches his hands; has fallen. *Intelligence.*—Difficult to attract his attention. Does not feed himself. Can drink from cup but allowed a bottle (now able to take milk). Restless at night. Self-willed. *Gait* unsteady, runs from side to side, head lowered.

A Case of Congenital Malformation of the Heart.

By Donald Hall, M.D., F.R.C.P.

The patient, a girl, aged 4 years, is deeply cyanosed and has clubbing of the fingers and toes. There is a history of fits. A systolic murmur is present over the entire precordium, with maximum intensity in the pulmonary area; there is also accentuation of the second sound. The electro-cardiogram shows right-sided preponderance and increased amplitude of the initial ventricular complexes. Blood-count: red cells, 7,200,000; hæmoglobin, 148 per cent.; colour index, 1.



A Case for Diagnosis.

By FLORENCE EDMONDS, M.D.

H. W., AGED 6. Full term baby, weight 7½ lb. Normal confinement. Suffered badly with eczema, and difficult to feed, otherwise normal, and development proceeded normally. A bright child, but very disobedient. All functions normal. Speech normal and able to say alphabet and to count.

April, 1926.—Three months' excitement. Voluntary speech gradually ceased but patient answered questions.

July, 1926.—Gradually became better, had laughing fits, but not speaking at all. Not sleeping well. Bowels constipated, appetite fair.

September, 1926.—Very restless again, running up and down room. Appetite enormous and all thought seemed concentrated on food. Answered questions occasionally, but very indistinctly. Sleeps better. Bowels regular. Since then has gradually become vindictive to other children, but in other ways has improved. He will sometimes sit up and play with his toys quite intelligently. His appetite is normal and he is sleeping well.

Specimen: Kidneys obtained Post Mortem from a Case of Pyelonephritis.

Shown by EVELYN SCOTT, M.D.

R. J. F., AGED 6 months. Admitted June 21, 1927. Child had been sick and fretful for ten days. On admission there was marked meningismus, with a high temperature. The fontanelle was depressed and the cerebro-spinal fluid was not under pressure. Both kidneys were easily palpable. The urine contained abundant *Bacillus coli* and pus cells. Death four days after admission.

A Case of Gangrene of the Leg and Arm.

By WALTER BROADBENT, M.D., F.R.C.P.

A. D., AGED 2½ years, admitted April 7, 1927. The child had not been looking well for two or three weeks. The day before admission he complained of a pain in his right leg when running about.

Next morning his eye and his leg were black and he was brought up to hospital.

The right eye was markedly proptosed and subconjunctival hæmorrhage obscured any white area. There was a large black swelling of the right side of the face from the eyebrow to an inch above the mouth. The right leg and foot were black from about 4 in. below the knee.

Temperature 99·6° F, pulse 120, respiration 26. The child took food and did not seem nearly as ill as one would have expected.

Two days after admission the left arm began to become black, and, in a few hours, there was well-marked hæmorrhage under the skin of the left forearm and hand, which were much swollen.

Blood-count.—Red cells, 4,770,000; hæmoglobin, 40 per cent.; leucocytes, 6,000; polymorphs, 65 per cent.; lymphocytes, large, 15 per cent.; lymphocytes, small, 20 per cent. Wassermann reaction negative.

The heart sounds were good; no murmur suggesting possibility of emboli. Lungs, abdomen and kidneys showed no abnormality.

The condition as seen in the left arm looked like extensive subcutaneous hæmorrhages spreading rapidly towards the hand.

Temperature round about 99° F. for the first week. Later, when gangrene was progressing, it became hectic, rising to 101° F. or 102° F. at night.

The swelling of the face cleared up in about three weeks, without any breaking of the skin. The limbs were kept dry in cotton wool, and allowed to separate without any surgical interference. The hand and wrist came off in about seven weeks, and the foot and portion of leg somewhat later. The stumps are, of course, not satisfactory, and they will want trimming when he is a little older.

I am unable to offer any explanation of the cause of the original illness.

Three Cases of Osteomyelitis.

By J. R. Griffith, F.R.C.S.

Case I.—V. H. *Osteomyelitis of Femur followed by Endocarditis.*—On February 20, 1924, he ran into a door, bruising his left knee very slightly. The knee became painful next day, and three days later the temperature rose to 100° F., but there were no serious local symptoms. On February 25, five days after the accident, tenderness was noted over the lower femoral epiphysis, extending up the shaft of the femur. On exploration of the bone subperiosteal pus was found half way up it, the outer half of the circumference of the shaft was therefore removed from the lower three-quarters of the bone. The boy did fairly well until July 22, 1924, when a sequestrum, about 6 in. long, was removed. Immediately after the operation he became very ill, all the other joints became painful, the heart dilated, and evidence of vegetations on the mitral and aortic valves was noted. After remaining very ill for about four months, he began slowly to improve. The wound healed the following March, and he was discharged two months later.

Case II.—Osteomyelitis of left Tibia.—On July 24, 1925, the child (H. P.) fell against a large stone on the beach, within a few hours the leg became painful, the temperature rose, rigors set in and he became delirious. Admitted on July 28 and immediately operated upon. The periosteum was separated from the shaft by pus in the upper half of the tibia. A subperiosteal resection of the shaft was carried out. Bone regeneration proceeded at the upper and lower end, but tailed off in the middle, leaving a gap.

On January 4, 1927, a further operation was done in order to give him a stable leg. The ends of the tibia were shaped so as to form a morticed joint, and a segment of the shaft of the fibula was removed so as to allow the shortened ends of the tibia to come into contact. Union took place satisfactorily, and two months later the boy began to walk.

Case III.—E. I. *Osteomyelitis of Left Tibia.*—On August 21, 1924, patient (a boy) developed swelling and pain over the lower end of the left tibia without any apparent cause; he had had diphtheria eight years before, followed by a mitral systolic murmur. Over the lower end of the tibia the periosteum was found separated from the shaft by pus, and pus was oozing from a sinus in the bone just above the epiphysis. About 6 in. of the lower end of the shaft were therefore removed subperiosteally. Regeneration took place rapidly, the wound was healed in four months, and in the following March the skiagram showed a very good new shaft.

A Case of Infantilism of Rheumatic Origin.

By G. L. Lyon Smith, M.R.C.P.

P. W., GIRL, aged 7 years.

History.—Pyrexia with swollen knees, wrists and phalangeal joints (fusiform). Treated by salicylates with marked improvement. Has remained about the same weight for the last two years.

Present Condition.—Very small. Some swelling of wrists and fusiform swellings of phalangeal joints. Conjunctivitis and iritis.

Bacteriology.—Fæces: no excess of streptococci, no abnormal *Bacillus coli.* Urine: sterile; tonsils ?.

Treatment.—Salicylates; rest in bed; tonics—cod-liver oil and malt; anterior pituitary gland and thyroid (Gardiner Hill).

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Case of Ethmoidal Suppuration.

By M. FITZMAURICE-KELLY, F.R.C.S.

L. A., MALE, aged 7 years, sent into hospital with proptosis of two days' duration. Temperature 100·4° F. Definite redness and œdema of lids, not of conjunctiva. Diagnosis of orbital cellulitis made, and the orbit explored, without, however, pus being found. Four days later he was seen by Mr. H. H. Taylor, who reported that such cases were due to ethmoidal suppuration, and that exploration of the inner wall of the orbit would reveal pus. This proved to be correct, and the boy has progressed well since.

As the fact that ethmoidal suppuration is a relatively common cause of orbital suppuration does not seem to be generally known, it appears worth while to record this case.

A Case of Anterior Poliomyelitis.

By WALTER BROADBENT, M.D., F.R.C.P.

M. V., GIRL, aged 3½ years. Admitted March 26, 1927. First ill about a fortnight before admission with a bad cold, and a few days later her doctor found some crepitations in the left upper lobe. Ten days later she vomited and complained of headache and became irritable. A squint was noticed the next day and she was delirious at night. When seen on March 26 she was very drowsy, the left arm was weaker than the right, the left chest moved much less than the right in respiration. She complained of pain when the head was pulled forwards, crying when the chin was made to reach the chest. She was very thin, had a retracted abdomen, and Kernig's sign was present, though not marked. Toe reflexes flexor. No squint could be seen, but when asleep her eyes were half open. She presented a picture very like a commencing meningitis. On lumbar puncture the fluid was clear and not under pressure. It contained a trace of albumin. Fehling's solution was reduced normally. Cells were 15 to the c.mm. Lymphocytes 92 per cent. Polymorphs 8 per cent. Hexamine was administered. In the course of the next week or two the child slowly improved in general condition but paralysis of the left shoulder girdle became more marked, and the head began to lob over, showing the paralysis of the neck muscles. The difference in movement of the two sides of the chest only lasted for forty-eight hours. It was then evident that it was not a meningitis but a poliomyelitis of the cervical region (first to fifth nerves). The difference in breathing was due to implication of the roots of the phrenic nerve. A splint to hold up the arm and to fix the head was made by the house physician, Dr. Rush, and though wasting of the muscles involved became more evident for a few weeks, there has since been a slow but steady improvement.

Case of Lateral Spina Bifida.

By M. FITZMAURICE-KELLY, F.R.C.S.

I. W., A FEMALE child, aged 4 years and 4 months, had been brought to the out-patient department when 1 month old with a swelling of the left buttock, and apparently some shortening of the left lower limb. The swelling was cystic, and was situated beneath the gluteus maximus muscle. There was a dimple in the skin overlying it. Later X-rays showed a deficiency of the lateral mass of the sacrum and of the fifth lumbar vertebra. The case had been kept under observation for a time, and the child learnt to walk, though with a limp. She had then been lost sight of for a time; about a year previously the mother had brought her up with severe paralytic talipes of both feet and incontinence of urine. She had been walking on the dorsum of the right foot, and the deformity was so severe as to necessitate an osteotomy. She now walked fairly well in an apparatus, and should she reach the age of ten a stabilizing operation on the feet ought to make her fairly comfortable.

74 Scott: *Dextrocardia;* Griffith: *Hirschsprung's Disease*

A Case of Dextrocardia.

By EVELYN SCOTT, O.B.E., M.B.

ADMITTED for malnutrition and attacks of cyanosis. On examination there is seen to be well marked dextrocardia, with marked enlargement of the heart and almost complete airlessness of the right lung. The liver is just palpable and is on the right side. Radiogram shown.

A Case of Lesion of the Corpus Striatum.

By EVELYN SCOTT, O.B.E., M.B.

P. J., MALE, aged 9 years. Admitted June 29, 1927. Full-time child, normal confinement, weight at birth 9 lb. Second child of four, all the others being well. Sat up at 10 months. Fed on breast for 9 months. Not backward in talking.

Previous History.—"Pneumonia" at 18 months. Ill for one week. Bronchitis and whooping cough at 8½ years. Went to school when he was 5 years old, and did not in any way appear abnormal. After he had been to school a short time he began to fall on the back of his head several times in the morning. This went on for six months, until his mother took him to see a doctor. She was advised to take him to Lady Chichester Hospital "for his nerves." At that time she did not know to what to attribute the falling, but in view of subsequent events she thinks he fell because of some weakness in his legs. He was four months in Lady Chichester Hospital, when he was removed because he was burned under the knee (probably by a hot water bottle). When he came out he could not use the leg that had been burned. A short time afterwards his mother noticed that the left arm was twisted up, and it became apparent to her that the boy was more or less paralysed. He was then taken to Hove Hospital, where he received massage and radiant heat treatment for eighteen months. He improved under this treatment, but six months ago his mother was not able to take him to the hospital, and since that time he has retrograded to his present state. The mother now complains that he cannot sit up, cannot walk, does not feed himself, cannot dress himself, and during the past three months he wets himself when he is out. He is forward in remembering and he talks quite well. On examination: there is no evidence of disease of the pyramidal system. There is well-marked "lead-pipe" rigidity.

Family History.—Unimportant, except that mother is "nervy."

A Case of Perforation of the Bowel in a Baby: Hirschsprung's Disease.

By J. R. GRIFFITH, F.R.C.S.

R. S., MALE infant, aged 6 months, admitted on March 16, 1927, suffering from severe abdominal pain and tenesmus; on examination a sausage-shaped lump was felt in the pelvis, a diagnosis of intussusception was therefore made, and the abdomen was opened under an anæsthetic. A section of the pelvic colon was found to be œdematous, thickened and immobile, it pitted on pressure and seemed absolutely lifeless. At one point low down in the area the bowel was perforated with a transversely disposed fissure, through which pale fæcal material was leaking. A tube was therefore tied into the bowel and colostomy performed by the glass rod method. The child unexpectedly recovered and has developed into a very healthy baby.

Case of Torticollis.

By M. FITZMAURICE-KELLY, F.R.C.S.

E. H., FEMALE, aged 9 years, showed a virtually perfect result. The original deformity had been severe, and there has been considerable secondary scoliosis.

COMMENTS.

The following points required emphasis.

(1) *Operation.*—He (the exhibitor) preferred an open division of all tight structures, including the fascia deep to the sterno-mastoid, down to the carotid sheath and sometimes the anterior fibres of the trapezius.

(2) *After-treatment.*—He used rigid fixation in plaster of Paris at the time of operation, in the most extreme over-correction which could be attained at the time. A window was cut to remove the stitch (subcuticular, of silkworm gut) and the apparatus was retained for six to eight weeks. Following its removal, massage and active exercises were begun. He (the exhibitor) believed the results were better than in cases where no retentive apparatus was used. A subsidiary advantage was that the duration of treatment in hospital or nursing home need not last more than a few days.

Case of Ventral Hernia.

By M. FITZMAURICE-KELLY, F.R.C.S.

J. W., FEMALE, aged 10 years. A very severe ventral hernia, admitting four fingers in a line, following an operation for pneumococcal peritonitis. He (Mr. Fitzmaurice-Kelly) showed the case as the first one in which he had operated by Gallie's method of fascial or living suture, in February, 1925. Now, more than two years afterwards, the wall was sound, and the sutures and knots could still be distinctly felt under the skin. He had since given the method a fairly extended trial in cases of hernia which had relapsed or been rejected as unsuitable for operation. In his cases (forty-two in all) there had been no case of relapse so far. He was very much impressed with the value of the method.

Three Cases of Foreign Bodies Swallowed by Children.

By GEORGE MORGAN, F.R.C.S.

Case I.—Girl, aged 6 years. Admitted to the Children's Hospital on account of very severe stomatitis, and threatened cancrum oris. She died quite suddenly two hours after admission. At the post-mortem examination a farthing, covered with cupric acetate, was found fixed in the œsophagus, having ulcerated through the œsophageal wall into the left innominate vein and caused death by hæmorrhage.

Case II.—Patient, also a girl, aged 6 years. She had a constant cough accompanied by a large amount of blood and muco-pus which she spat up, often amounting to two-thirds of a teacupful in the day and night. Her temperature was hectic and her pulse rapid. There were signs of consolidation and cavitation at the right base. Several of the staff of the Royal Sussex County Hospital who saw the child diagnosed a basic cavity and gave a hopeless prognosis. There was no history of foreign body and it was before the days of X-rays. The case was under observation for three months and the patient seemed to be rapidly getting worse. About this time she began to cry out, complaining of a pain in the rectum, and on examination I found and removed a button shank from the bowel. It had been covered with some composition to imitate pearl, but this had been destroyed in the body, only the flat piece of metal and the eyelet remaining.

When the child's mother was questioned, she remembered that she had had a choking fit some six weeks before she became ill. She (the mother) took her into a chemist's shop. The chemist slapped her back sharply and said that she would be all right.

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The button removed from the rectum had doubtless caused the choking fit and been inspired into the trachea and bronchi, eventually producing a lung abscess. This had gradually ulcerated through and discharged into the digestive tract, the foreign body being discharged along with the pus.

The child lived, and after a long convalescence became perfectly well.

Case III is unique in my experience. Patient, a little girl, aged 1 year and 10 months, was seen in consultation with Dr. Simpson. She complained of a swelling at the angle of the jaw which had formed and softened in three days. The swelling was as large as a walnut and showed obvious fluctuation.

It was incised under chloroform and evacuated. Several small feathers came out in the pus at the time of the operation, and three more escaped in the discharge during the next ten days. The child had been noticed pulling feathers from her eiderdown pillows, and swallowing them. There seemed to be little doubt that some of the feathers had entered the tonsillar crypts and passed through into the cellular tissues of the neck, causing the abscess.

Dr. MURIEL L. M. NORTHCOTE showed Two Cases of Nervous Disorder.

Mr. J. R. GRIFFITH, F.R.C.S., showed a Case of Slipped Epiphysis of the Femur.

Section of Dermatology.

President—Dr. J. H. SEQUEIRA.

Early Angioma Serpiginosum, confined to One Shoulder.

By F. PARKES WEBER, M.D.

THE patient, C. L., aged 24, has about ten small superficial cutaneous lesions, which have gradually developed during the last two years over the right shoulder, in the region of the supraspinous fossa. Otherwise she seems to be a well-developed young woman, healthy in every way. The individual lesions are scarcely, if at all, raised, are red or red-brown in colour, and are not accompanied by any itching or sensory disturbance. She says she would not have known they were there had not she and her mother seen them. Some of them are speckled with small pin-point red telangiectases. The older lesions are browner and slightly shiny. The earliest lesion, which is of two years' duration, is about as large as a farthing.

Comparing this case with a very similar one (in a young woman aged 21½) that I showed at the meeting of March 17, 1927, in which the lesions were confined to the right upper limb, and with a case of longer duration shown on May 19 by Dr. G. B. Dowling, I have little doubt that it is one of early so-called angioma serpiginosum, though the lesions are perhaps slighter than in any case previously recorded. I am indebted for the case to my colleague, Dr. E. Schwarz, who was struck by the resemblance of the lesions to those in my above-mentioned previous case. Probably the slighter forms of angioma serpiginosum are not so rare as is generally supposed.

Although in cases of angioma serpiginosum a certain amount of pigmentation of the lesions occurs, I do not think that pigmentation is ever seen comparable in degree to that in Schamberg's disease—and this is a reason why angioma serpiginosum should be distinguished from Schamberg's disease, which latter appears, moreover, to be almost as confined to the male sex as the former seems to be to the female sex (remarked on by Dr. J. H. Sequeira).

In slight and only slowly progressive cases, like the present one, it is doubtful whether treatment is advisable even for cosmetic purposes.

[The case of A. E. F. (a young woman, aged 21½), previously shown by Dr. Weber at the meeting of the Section on March 17, 1927, was again shown, for comparison with the above case. The Members present apparently agreed that both cases were examples of "angioma serpiginosum," in spite of the limited extent of the lesions.]

Discussion.—Dr. J. M. H. MACLEOD said he saw one of the cases, that of the girl, six months ago, and regarded it as a mild example of the condition. Various treatments had been tried without success.

Dr. J. H. SEQUEIRA (President) asked whether any Member had seen such a case in a male?

Dr. WEBER said he did not remember having seen accounts of angioma serpiginosum in males, unless indeed some of the cases described as examples of Schamberg's disease in males were really of the same nature as angioma serpiginosum.

108 Dowling: *Acne Agminata; Rare Seborrhœide*

Case of Acne Agminata.

By G. B. DOWLING, M.D.

M., AGED 27. Sailor. Duration of eruption about eight months.

The eruption consists of a large number of scattered, mainly discrete, reddish-brown papules, varying in size from that of a pin's head to that of a millet seed. They are present on the face, chin, nose, forehead, and both upper and lower eyelids. Here and there the lesions are closely aggregated and form small, almost confluent, masses of papules. Individually they resemble isolated nodules of lupus. Some have involuted, leaving small yellowish-brown pitted scars; these also have an opalescent appearance.

The von Pirquet test has been applied and the result is completely negative.

The patient's general health is excellent.

Case of Rare Seborrhœide.

By G. B. DOWLING, M.D.

M., AGED 38. Labourer. Duration of eruption, eight months.

The condition began somewhat suddenly, while the man was apparently in a condition of perfect health, with a profuse eruption of papules and nodules of various sizes all over the face, chin, nose, forehead and beard-bearing region of the neck. The eyelids were spared except for two small spots, one on each upper lid. The lesions were so densely crowded together as to present a practically confluent eruption. The face appeared congested, but on examination the individual papules and nodules were found to vary in colour from pinkish-yellow to almost white. Some of them resembled infected sebaceous retention cysts almost ready to break down, but in reality they were quite solid, and at no time has there been any tendency to pustulation.

The patient has been treated with small doses of X-rays and sulphur lotion locally. From the commencement of treatment there has been a steady improvement. All the lesions have become quite small, and with this involution there has been a marked alteration in colour, from opaque pinkish-yellow or white to opalescent yellowish-brown.

I gather from several senior Members here to-day who saw the original case described by the late Dr. Pringle as a "rare seborrhœide," that I am wrong in regarding this case as belonging to the same group, but my impression is shared by others, including Ketson who regards rare seborrhœide as resembling acnitis in many respects.

What I would like to point out is that although widely different to begin with, the lesions of both of these cases are now identical, only in one case they are far more densely crowded together than in the other and a much wider area is involved. The diagnosis of acne agminata is therefore presumably the correct one.

I would suggest further that neither of these cases is tuberculous in origin, in spite of the resemblance of the lesions to lupus nodules. In fact it appears that there is little evidence that acne agminata is ever tuberculous in origin. I am unable to find a reference to a case in which positive evidence of tuberculous origin has been produced. I, personally, think that coccal infection is capable of producing identical lesions; thus in some cases of rosacea with acneform lesions a certain proportion of the individual lesions bear a close resemblance to the lesions of acnitis,

exhibiting the characteristic yellowish-brown opalescence on vitro-pressure, so that at times one may be in doubt whether to call the case acne agminata or rosacea. I do not wish to suggest that rosacea and acne agminata are related, only that a lupoid nodule is not necessarily a tuberculous lesion.

Discussion.—Dr. J. H. SEQUEIRA (President) said these cases were well worth discussion. He had been appealed to by some of the younger Members for some guidance as to the classification of this group of agminate acneform lesions.

Dr. S. E. DORE said that many years ago he had shown a case which the late Dr. Pringle had stated to be an example of this rare seborrhœide. The patient was a woman, and the lesions were more discrete; there was no scarring, but rosacea was present, and there was a brownish infiltration on pressure.

Dr. W. JENKINS OLIVER said that the histological picture of the one case, which he had investigated and reported, showed a definite tuberculous structure, while inoculation into guinea-pigs gave a negative result for tuberculosis.

Dr. A. WHITFIELD said that the lesions, in cases of this kind, were found especially on the nose. He thought these were enlarged sebaceous glands; they were fatty and protoplasmic structures, and therefore liable to exhibit a translucent appearance, such as was seen in lupus nodules, especially if they were surrounded with a mononuclear infiltration. He did not think that was the explanation of the nodules in the present case. On sections being cut a marked granulomatous infiltration would probably be found. He believed the late Dr. Pringle's case had shown an enlargement of sebaceous glands, surrounded by a somewhat imperfect chronic inflammatory reaction. He did not consider that either of the cases shown had a relationship to seborrhœa or to rosacea. His view was that both were tuberculous, but the separation of nodular sarcoid from tubercle was difficult.

Dr. J. M. H. MACLEOD referred to Radcliffe Crocker's cases of acne agminata, and the stil more marked case of Dr. Galloway's, a picture of which was made and sections cut. In these cases the lesions were yellowish, small, translucent nodules, in groups, all over the face, and situated on a slightly inflamed base. Any suppuration present was secondary. Section showed a tuberculous-looking structure, but he believed there was no evidence of tuberculosis in any of the cases. The milder of Dr. Dowling's two cases looked like a follicular tuberculous condition of the type of acne scrofulosorum.

Dr. A. M. H. GRAY said he had been interested in this kind of case for some years, but he himself had seen very few of the condition, and he had not had a case of it under his own care since the war. Undoubtedly, as admitted by Radcliffe Crocker, the cases described by Barthelemy as "acnitis" and those described by Crocker himself as "acne agminata" were the same. A true "acne scrofulosorum" could also occur on the face. Some years ago he (Dr. Gray) had shown a child, a patient of Dr. Still, who had typical acne scrofulosorum, extensively distributed on the limbs and well marked on face and scalp, causing scarring and, in the latter site, alopecia. In the Crocker and Barthelemy type the well-defined nodules were almost as large as lentils, and were dome-shaped and translucent. They rarely showed suppuration, though occasionally there was pus—probably secondary—in the follicle. On section there was no sign of necrosis, but a heaping up of tuberculous nodules between masses of fibrous tissue, just as in sarcoid. For years he had thought these were of sarcoid form, but his views had been slightly modified, because they did not fit in well with Schaumann's criteria, for every now and again there was a positive reaction to the tuberculin test. Schaumann maintained that the acne agminata of Crocker was the same condition as that which Continental dermatologists still called the "follicular lupus of Tilbury Fox," and if one of these dermatologists were now present he would probably make that diagnosis in this case.

Arndt, in 1914, had shown the speaker some sections taken from a case of this type, and there were many tubercle bacilli present in the section; but in the majority of cases of acnitis and acne agminata no tubercle bacilli could be found. The difficulty of the subject was recently shown in papers from America. Schamberg was strongly of the opinion that

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the condition was not tuberculous, whereas Maloney had investigated the matter and was convinced that it was tuberculous. Perhaps if all the published cases could be collected and thoroughly examined a conclusion might be arrived at, as no one person met with a sufficient number of cases to enable him to come to a conclusion on his experience alone.

Dr. DOWLING (in reply to Dr. Whitfield) said that Dr. Milian had shown cases of rosacea, the lesions of which resembled lupoid nodules, to the French Dermatological Society. Sections were exhibited which showed that the structure of a nodule was that of a granuloma, with giant cells and epithelioid cells. No connexion with the sebaceous apparatus was demonstrated, but it was suggested that if serial sections had been taken through the whole lesion the relation of the infiltration to a pilosebaceous follicle might perhaps have been demonstrated.

Case for Diagnosis.

By G. B. DOWLING, M.D.

AGED 50. Railwayman, in otherwise good health. Duration of condition twelve years ; lesions have become more widespread in the past few years.

Condition is a generalized patchy erythrodermia with moderate infiltration of the skin in some places, and well-marked infiltration in others, especially on the thighs. Islands of normal skin are found here and there in the erythrodermic patches.

There appears not to have been much irritation. Desquamation is not a marked feature at present.

The following investigations have been made.

White cell count, 14,000.

Differential Count on two occasions : Report by Dr. Bailey.

I.		II.	
Polymorphs	40 per cent.	Polymorphs	81 per cent.
Lymphocytes	31 ,, ,,	Lymphocytes	11 ,, ,,
Large mononuclears	17 ,, ,,	Large mononuclears	1 ,, ,,
Eosinophils	8 ,, ,,	Basophils	2 ,, ,,
Basophils	0 ,, ,,	Myelocytes	5 ,, ,,
Myelocytes	1 ,, ,,	Both films show considerable numbers of large cells between large mononuclears and large lymphocytes ...	
Polymorph leucopænia with increase in large mononuclears and eosinophils			

REPORT BY DR. J. M. H. MACLEOD.

(1) *Section from a Comparatively Non-infiltrated Patch.*—This shows considerable corrugation of the surface of the skin. There is hyperkeratosis and much overgrowth of the prickle-cell layers with œdema. The downgrowth is irregular and numerous small islands of corium are shut off between processes of the prickle-cell layer. There is a profuse cellular infiltration of the upper part of the corium, having a fairly clear line of demarcation. The cells are small, spherical or fusiform in shape. Similar cells are clustered about the capillaries. There is no evidence of destruction of the corium nor are any cells described as peculiar to mycotic conditions found.

(2) *Section from a more Infiltrated Patch on the Front of the Right Thigh.*—In this section there is in the infiltration a fair number of large fusiform or oval cells, with granular protoplasm and a large nucleus. A few of these cells are crenated, and there is fusion here and there of two or three cells to form something like a giant cell.

On the whole, however, the infiltration cannot be described as dense; it is, in fact, less profuse than in the first section.

My original diagnosis was the prefungoid stage of mycosis fungoides, but in view of the comparative absence of itching, the lack of response to X-ray treatment and the histology, it is difficult to adhere to this view.

On the other hand, the degree of infiltration appears to be too great for any variety of parapsoriasis, and the infiltration of cells in the corium also appears somewhat dense for this condition, though hardly sufficiently dense for a premycosic eruption. The blood-counts do not suggest the type of case described by Dr. Sequeira as lymphoblastic erythrodermia.

Postscript, July 1, 1927.—The condition appears now to be showing some response to X-ray treatment; one may, in fact, assert that there has been definite improvement.

Bowen's Disease.

By J. H. T. DAVIES, M.B., B.Ch.

I AM showing this case for two reasons; first, because I have the impression that Bowen's disease is not generally regarded as having a distinct identity, and secondly, to show the effect of X-rays on a lesion which elsewhere has been described as only suitable for surgical excision. I think if you will compare this case with the two first cases described by Bowen in the *American Journal of Cutaneous Diseases*, xxx, p. 241, you will agree that the striking similarity between these cases entitles the condition to be regarded as a distinct clinical entity.

My patient is aged 55, and has had a single group of lesions on the outer side of her knee for twenty years. There is no family history of skin disease. It was a plaque of leathery consistency, 5 in. by 4 in., with a sharp circinate border studded with round or oval, dome-shaped papules averaging 1 cm. in diameter, some discrete and some confluent. The surface was warty and covered with crusts. In the centre was an ulcerated mass of granulations exuding pus. When the crusts were removed there was some tendency to bleed, but the colour of the lesions was a yellowish brown. There was no cicatricial tissue. The lesion has since been treated with X-rays, eight weeks ago 4 H unfiltered, three weeks later 6 H/1mm. aluminium. The proximal part of the lesion has healed as the result of this treatment without leaving a scar. I have no doubt of the efficacy of the rays. I had originally intended to have the lesion destroyed by diathermy, and while she was waiting for admission to hospital I had ample opportunity of trying various forms of local treatment. The first dose of X-rays cleaned the lesion, and I expect the third or fourth to remove it altogether.

As regards the histology, a piece was taken from the edge of the lesion. There is parakeratosis and hyperkeratosis, the strata lucidum and granulosum are absent. The prickle-cell layer is enormously hypertrophied, twenty-five to fifty layers, the superficial cells are swollen with a clear space round the nucleus, and the protoplasm is fibrillated and has undergone partial horny degeneration. In the middle part of the epidermis are:—

(1) Normal cells.
(2) Cells with abnormally large nuclei.
(3) Cells with two or more nuclei.
(4) Groups of cells with deeply staining protoplasm and faint nuclei.
(5) Many cells in active mitosis, sometimes normal and sometimes abnormal.
(6) Concentric groups of cells undergoing horny degeneration and resembling "cell-nests."
(7) Masses of protoplasm containing several small but well-formed nuclei.

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(8) Cells distended with œdema and nucleus pressed against the side in some cases, and lying shrivelled in the middle in others. I did not see any cells analogous to the "corps ronds" of Darier. There is not much general or intercellular œdema of the epithelium. The basal layer is normal and intact and is conspicuous in contrast with the Malpighian layer by the rarity of the mitoses seen.

The papillæ are œdematous, the capillary is greatly dilated; there is very slight cell infiltration. In the papillary layer there is slight cell infiltration round the vessels, mainly lymphocytes (no plasma cells and scanty fibroblasts); e.g., 100 cells to the

average vessel. There is œdema of the corium, but the connective tissue is not markedly degenerated, and the elastic fibres are stainable. Round the vessels in the subpapillary layer are seen small collections of plasma cells.

Case of Leucoderma.

By M. SYDNEY THOMSON, M.D.

PATIENT, C. B., female, aged 21, first attended the out-patient department at King's College Hospital two days ago. Her history may be briefly summarized as follows. Eleven years ago she noticed pigmentary changes in her skin. Sir Malcolm Morris diagnosed leucoderma and the disease progressed until loss of pigment became complete three years ago, the last areas to become white being the knuckles. When the affection first started the patient also suffered from severe Graves' disease. This is still present although it is now much ameliorated. It is of interest that three of the patient's maternal aunts also have leucoderma. Some doubts have been expressed that the disease is here present at all, but I would like to point out that the history of early reddening after exposure to the sun and complete absence of pigmentary reaction are sufficiently typical to confirm the diagnosis. At the end of last summer the patient noticed the appearance of brown patches on the anterior surface of the lower third of the legs. These are only faint macules, varying in size from that of a pin's head to that of a threepenny piece, but each is circular in outline and presents no concave erosions such as would be seen if these areas were the remains of an advancing leucoderma. I have brought the patient in order to obtain the opinion of the Section as to whether these represent an abortive attempt at freckle formation or whether it is possible that they represent the first signs of recovery of function on the part of the skin.

Discussion.—Dr. J. H. SEQUEIRA (President) said that the association of Graves' disease with leucoderma was one which had not been sufficiently recognized. He recently saw a case of well-marked leucoderma associated with Graves' disease, and in that patient the Graves' disease was still active. He had been waiting to see whether, when the general disease had improved, the skin condition would participate in the improvement.

Dr. PARKES WEBER said he thought that when vitiligo occurred in association with Graves' disease the pigmentation was usually more striking than the leucodermic patches. But in the present case practically the whole skin had become leucodermic. Surely this must be very rare, especially as the hair of the head seemed not to be in any way affected.

Dr. M. SYDNEY THOMSON (in reply) said he agreed that one would naturally expect to see more affection of the hair; but there were a large number of white hairs in the scalp and about the body generally.

Case for Diagnosis. ? Lupus Erythematosus.

By M. SYDNEY THOMSON, M.D.

L. B., FEMALE, aged 29, first came into hospital at the beginning of June, 1927. She states that two years ago she first began to notice the appearance of spots on the chest and back, each one having persisted ever since. On the chest they are all within an area the limits of which can be defined by lines drawn from the

xiphisternum to the outer ends of the clavicles. On the back they extend from the base of the neck to the coccyx, but are all within 2½ in. of the mid-line. They vary in size from that of a pea to a shilling. Each has a definite circular or oval outline and is pinkish in colour with a faint suggestion of yellow. Diascopy leaves the diffuse yellow tinge. Here and there the centre of the lesion is of a deeper red and conveys the suggestion of erythema multiforme when seen from a distance. There is a scale on each of these flat papules which leaves a moist area on removal. Occasionally the removal of the scale is accompanied by the tearing of a triangular piece of epidermis from the apparently normal skin surrounding the lesion. This occurs in much the same way as in a hang-nail upon removal. The patches on the face and scalp appeared three months later. That on the scalp is irregular in outline, roughly of the size of a sixpenny piece and is covered by fine, cretaceous and closely adherent scales. On the face, the suborbital region of each cheek only is affected. Here much the same appearances are presented as by the lesions on the back, but there are the following additional points to be noted. There is definite pitting, and this is mostly due to the scaling, for the removal of the offending scale allows the underlying tissues to expand and occupy their normal position. In one place, however, the pit is due to a definite but fine and atrophic scar. The scales are not cretaceous, but are very tough whilst yet retaining pliability. They are moderately adherent and occasionally show plugs on their under-surfaces. There is nothing else of importance in the history of the patient or in the appearance of the lesions themselves. Pathological investigations are not yet complete. The suggested diagnosis is that of lupus erythematosus.

Case for Diagnosis.

By A. M. H. Gray, C.B.E., M.D.

Patient, a male, aged 44, apparently in perfect health. The present eruption began some two years ago as two red patches on the outer part of the left thigh. These have grown till they have reached their present size and condition; three more lesions have also appeared since the original onset. The last only appeared about two months ago.

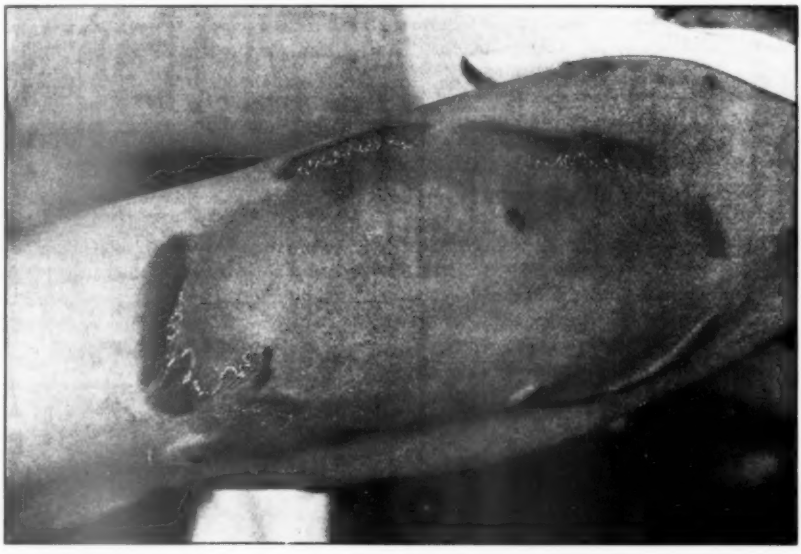
The distribution of the lesions is shown in the accompanying photograph. An oval area some 9 in. by 5 in. is incompletely surrounded by a raised margin some ½ in. to ¾ in. in width and raised about ½ in. from the skin surface. The middle of the posterior rampart is incomplete. In the centre slightly atrophic pigmented areas are found enclosed by the upper and lower parts of the oval margin, the centre being clear; this suggests that the main oval lesion is formed by two primary patches which have partially coalesced. The edge is pale pinkish-brown in colour, is semi-translucent and shows distension of the follicular openings.

Another similar patch is present behind the left knee towards its outer side; this patch measures 4 in. by 1½ in., and has a raised margin only in the upper and lower parts.

A third lesion, taking the form of a dome-shaped elevation, is present behind the lower part of the larger lesion and this is surmounted by a definite deep-seated bulla.

There are also present one or two scattered, slightly infiltrated erythematous patches in the neighbourhood of the larger lesion and also one on the outer side of the right hip.

My first view of the case was that it was a keloid, possibly following a serpiginous syphilide. The Wassermann reaction is, however, negative and there is no clinical evidence of syphilis. The section under the microscope shows an ill-defined cellular exudate, containing numerous plasma cells, but the most striking feature



consists in the large number of giant cells present; these are scattered about without any definite arrangement.

In view of these facts, I am inclined to place the condition in the group of chronic persistent erythemata: possibly allied to granuloma annulare or to the condition Crocker has named "erythema elevatum diutinum."

Discussion.—Dr. S. E. DORE said he thought this was a case of mycosis fungoides. The crescentic infiltrated patches, spreading from a central lesion and undergoing involution without scarring, and the presence of itching seemed to him to be characteristic of this disease.

Dr. J. M. H. MACLEOD said he did not agree with Dr. Dore's view, and thought that granuloma annulare should not be ruled out. He had made sections from cases of granuloma annulare, which showed a very dense, granulomatous infiltration, with some plasma cells, but no giant cells. The small upper lesion in the present case suggested granuloma annulare.

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Dr. J. H. SEQUEIRA (President) said he could not place the case, but he was not inclined to call it granuloma annulare, owing to its dimensions and its thickened border. He asked that a further report be supplied at a future meeting.

Dr. GRAY (in reply) said that he had always maintained that there was a chain of relationship between the more acute erythema multiforme on the one hand and the more chronic granuloma annulare on the other, and that there were a number of intermediate groups. The present case, he considered, belonged to an intermediate group. It was a very unusual case, and he had never previously seen anything like it.

CORRIGENDUM.

The following reply should have followed the remarks in the discussion on Dr. Savatard's paper, "Ichthyosis and Cancer of the Skin." See *Proceedings*, 1927, xx, 1596 (Sect. Derm., 100).

Dr. SAVATARD (in reply) said that what he had wished to demonstrate was that cancer of the skin (both basal and squamous) not infrequently arose on a pre-existing keratotic lesion: that in such patients an examination of the whole surface of the skin often revealed the fact that their skins were ichthyotic or xerodermatous—the ichthyosis was not necessarily marked. Further he had found that in several cases of cancer arising *apparently* on normal skin there was an associated ichthyosis.

He did not contend that oils and tar were not factors in the causation of cancer; he knew that they were, but he considered that the type of skin was not sufficiently taken into consideration in estimating their responsibility as determining factors.

In reply to Dr. Barber he said he had seen two cases of basal carcinoma associated with psoriasis and one case in which a squamous carcinoma had developed on a psoriasis plaque though the patient had never taken arsenic.

Section of Neurology.

President—Sir JAMES PURVES-STEWART, K.C.M.G., C.B., M.D.

CASES SHOWN AT THE CLINICAL MEETING (JOINT MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION WITH THE SECTION OF NEUROLOGY) JULY 27, 1927.

Case of Subdural Blood Cyst in Right Frontal Region located by Pneumo-radiograms.

By Sir JAMES PURVES-STEWART, K.C.M.G., C.B., M.D.

R. M. W., MALE, aged 18. At the age of 12 had cerebral concussion following fall on the ice. Four months ago developed causeless headache and diplopia. No diminution of visual acuity. Optic discs were seen to be slightly blurred. The only other abnormal sign detected was paresis of the right external rectus and of the left face. Cranial nerves otherwise normal. No sensory or motor abnormality of the limbs. Reflexes, both deep and superficial, were normal and symmetrical on the two sides. Cerebrospinal fluid contained 6 lymphocytes per c.mm., albumin = 0·02 per cent., glucose = 0·05 per cent., gold curve = negative, and Wassermann reaction = negative.

Optic discs became progressively swollen to 4·5 D. in the right eye and 2·5 D. in the left eye. Visual acuity and visual fields were normal, but the blind spot in both eyes was slightly enlarged. Pneumo-radiograms showed displacement of the right lateral ventricle, towards the left side.

In view of these findings, a wide decompression was carried out in the right frontal region. The subjacent dura was tense and, on being incised, a subdural, cyst about the size of a turkey's egg and containing black tarry blood, was evacuated. The subjacent cortex was then seen to be normal, except for brownish pigmentation of the pia arachnoid.

Clinical improvement since operation has been uneventful. The swelling of the optic discs rapidly subsided and there is now only a faint trace of left facial paresis. The bony gap on the cranium is concave.

A Case of Familial Periodic Paralysis.

By W. J. ADIE, M.D.

R. B., MALE, aged 13.

During the past four years has frequently awakened during the night or early morning to find that he cannot move hand or foot or speak. These attacks are unassociated with any regularly occurring previous event or experience which he has noticed. The weakness is maximal at its onset and passes off gradually—sometimes in three to four hours, sometimes not completely for two days. During the height of the attack he cannot speak or swallow and lies supine without any tendon responses and plantar-flexor plantar responses. These attacks occur on the average once each fortnight.

His father has suffered from exactly similar attacks since the age of four and they are much worse and more frequent than those of his son.

Myasthenia Gravis in a Boy Aged 10.

By W. J. Adie, M.D.

D. B., MALE, aged 10.

Rapid onset, two months ago, of double vision and generalized weakness of all movements, most severe in distribution of cranial nerves.

He now has a generalized weakness of all voluntary movements, most obvious in those of the face, eyes, jaw and neck, poor ocular movements, bilateral ptosis and slurred speech. There is no muscular wasting or fibrillation. The electrical reactions are normal in all muscles and the myasthenic reaction has not been obtained. The tendon responses are all much diminished and the plantar responses are plantar-flexor. On X-ray examination there is no evidence of enlargement of the thymus.

Congenital Deformity or Old Injury to Cervical Spine: Recent Onset of Paraplegia.

By W. J. Adie, M.D.

G. H., MALE, aged 36, a labourer.

Epilepsy since the age of 14. Left arm "always" smaller and weaker than the right. In his last fit he fell heavily on the back of his head: since this accident, whenever he hyperextends his neck, a feeling of painful tingling passes down his body to his feet.

Present Condition.—Gross deformity of spine in lower cervical and upper dorsal regions, fine nystagmus, bilateral ptosis, mild spastic paraplegia with extensor response on both sides, patchy analgesia over trunk and lower extremities, occasional incontinence of urine. Also has pterygium, accessory auricles, inguinal hernia and fibroid lung (left side).

Recovery, with Hemiplegia, from Vaccination Encephalitis.

By J. Purdon Martin, M.D.

D. L., FEMALE, aged 13.

Well till age of 5, then vaccinated. While the arm was bad she became very ill and was unconscious for about ten days. During that time lay very still. After recovery she had to learn to walk again. When, after two to three months, she returned to school she became "vicious"; had "terrible frenzies" if opposed, used to annoy other children and "pull their bows"; stole the school soap and other small things. After a few months she again became normal mentally, but it was noticed that she dragged her right leg. Since that time the right arm and leg have been weak, and the right arm has not grown as much as left.

Speech not affected by the illness.

Present State.—Mental development normal for her age. Right hemiplegia, with slight athetosis of fingers. No aphasia.

Microscopic slides illustrating vaccination encephalitis are on view.

Syphilitic Amyotrophy.

By DOUGLAS MCALPINE, M.D.

FEMALE, aged 53, married, three living children, three others died in infancy; two miscarriages.

Sixteen years ago patient noticed a weakness of her ankles; "the feet gradually dropped." This was accompanied by cramp-like pains in the legs. About thirteen years ago the left hand became weak; later the right hand was similarly affected. The muscles of the hands became wasted. There is no history of tabetic pains, diplopia or sphincter trouble.

Eyes.—Argyll Robertson pupils. No analgesia in centre of face.

Upper Limbs.—Weakness and wasting of extensors and flexors of both forearms and intrinsic musculature of both hands, left > right. Forearm muscles react to faradism somewhat feebly. No response to faradism in intrinsic muscles of either hand. Sluggish galvanic response. No sensory loss. Deep reflexes in the arms absent. No sensory loss in chest or abdomen.

Lower Limbs.—Double foot-drop. Complete loss of power of dorsiflexion of both feet with moderate atrophy of extensors of feet. All other movements of both legs quite good including plantar flexion. No other muscular wasting. No response to faradism, and sluggish response to galvanism in extensors of both feet. Vibration sense diminished in both legs. No other loss of sensation (especially, no loss to painful stimuli). Deep reflexes both legs absent. Plantar responses flexor.

Serological Findings.—Blood Wassermann: feeble +. Cerebro-spinal fluid: Wassermann reaction negative, cells 1-3 per c.mm. Total protein 0·05 per cent. Nonne-Apelt: negative. Lange curve, 0000111000.

Remarks.—A diagnosis of tabes dorsalis with amyotrophy is suggested by: (1) Argyll Robertson pupils; (2) absence of deep reflexes. Against this diagnosis is the absence in the history of tabetic pains and the conservation of pain sensibility. An amyotrophic syphilitic meningo-myelitis (Martin) would not account for the symmetrical wasting and in particular for the picking out of the extensors of the feet. The onset is suggestive of a polyneuritis.

Dyspituitarism with Double Optic Atrophy. (? Cause.)

By DOUGLAS MCALPINE, M.D.

BOY, aged 13, has been obese since birth. No increase in fat noticed for the last two or three years. At the age of 5 he found that his sight was defective in the left eye. His voice altered at the age of 12. He does not suffer from headaches or polyuria. History of convulsions in infancy.

Family History.—Father is a large man, and in his youth was very fat. Patient is the only child. No history of miscarriages.

Patient is a large, obese boy with a placid countenance. Mentally alert. Standard VII at school. There is an excess of adipose tissue all over the body, most marked in the face, breasts, hips, and abdominal wall. Sexual organs well

developed for his age. Pubic hair abundantly present, with feminine distribution. Weight, 9 st. 11 lb.

Eyes.—Pupils moderate in size, left larger than right. They react to light and accommodation. Ocular movements full; nystagmus to right. Almost complete atrophy left disc. Atrophy temporal half of right disc. Appearance suggests primary optic atrophy. Vision right $\frac{6}{9}$, and left $\frac{6}{60}$. Both visual fields show moderate general constriction, more marked on temporal side. Examination of the central nervous system is otherwise negative. No signs of congenital syphilis.

Serological Findings.—Blood: Wassermann negative. Cerebro-spinal fluid: Wassermann negative. Total protein 0·085 per cent., cells 1 to 3 per c.mm. Lange colloidal gold curve: normal. Blood-sugar curve: highest figure (after 50 grm. glucose) 0·148. Typical flat curve of hypopituitarism.

X-ray Examination.—Sella turcica within normal limits. Clinoid processes well defined. No calcified area seen.

A Case of Falling on Noise and Surprise.

By Gordon Holmes, C.M.G., C.B.E., M.D.

D. D., aged 14, comes from healthy stock in which there is no history of any affection similar to that from which he suffers. Both parents and most of his other relatives are large-boned and tall. There is no epilepsy or psychopathic taint in the family.

He has always enjoyed good health. When aged two and a half he had scarlet fever and has increased in weight since then. He is intelligent and has done exceptionally well at school.

At the age of five it was noticed that he was liable to fall, often without any apparent cause, but usually on hearing a noise. This tendency to fall became more pronounced when he was about eight or nine years old, and since then any loud sound, as a tyre bursting, a motor hooting, a door slamming, a rifle shot, etc., may bring about a fall. Less frequently a surprise, such as coming into a room which he had expected to find empty, being overtaken silently by a cyclist, a wasp or a butterfly flying suddenly in through the window, a cat jumping on to a table, or being touched from behind, may bring about a fall.

The element of surprise is, however, not essential; he has frequently fallen, for instance, on hearing a gas fire which he was watching being turned off, go "pop." In falling he has fractured his limbs repeatedly.

The falls are not preceded by any warning, and from his own account there is never any degree of loss or disturbance of consciousness; this has been confirmed repeatedly by observation. There is no change of colour and no vertigo or giddiness. His limbs suddenly flex under him and he falls flaccidly to the ground. He has lately noticed his left leg weak for about thirty seconds after certain falls, but he can generally rise at once without any assistance.

He has been treated with thyroid, pituitary and other organic extracts, and lately by bromide and luminal, without any benefit.

He is unusually tall for his age and is unnaturally fat, but he plays no games and takes little exercise. His skin is soft and rather hairless; hair distribution is still of the adolescent type, but his genital organs are well developed. Radiograms of his skull reveal a rather small sella turcica. His visceral organs and urine are normal; his systolic blood-pressure is 120 and the diastolic 80. The blood-sugar curve shows a slight delay in descent.

There is no evidence of organic nervous disease; his reflexes, optic discs and visual fields are normal.

On June 6, 1927, whilst he was in hospital he had an epileptic attack at 4.40 a.m. The onset was sudden during sleep; he became blue, his limbs were convulsed and his eyes deviated to the right. He did not lose sphincter control or bite his tongue, but he now admits that he has occasionally noticed a soreness of his tongue on awaking in the morning.

A Case of Crossed Tremor.

By Gordon Holmes, C.M.G., C.B.E., M.D.

L. B., aged 33, was admitted in July, 1927, to the National Hospital, complaining of tremor of the left arm and leg and twitching of the right side of her face.

Three years ago, within half an hour of a difficult confinement, she became tremulous all over, but the trembling ceased after an injection, the nature of which is not known. She had no disturbance of sleep, no drowsiness, no ocular palsies or other symptoms.

Two months after the birth of her child both arms and legs began to shake; the shaking of her right limbs lasted a short time only, but that of her left limbs persisted and became steadily worse till a year ago, when it became stationary. Fifteen months ago the right side of her face began to twitch and for a time she could not open her eyes. There was no disturbance of vision. During the past year her left arm and leg have been weaker and there has been some numbness of her hand and foot.

Past health has been good. She has been married five years and has had one miscarriage at ten weeks.

Present State.—Ptosis, which is variable and slight, of the right lid, and occasionally tonic spasm of the right orbicularis palpebrarum. There are frequently rhythmic movements of the right side of her face. Vision is normal and there is no diplopia: the fundi are also normal. Her tongue is tremulous, but the functions of her other cranial nerves are unaffected.

The right limbs are normal. The left limbs are slightly rigid and weak, and are involved in a coarse irregular tremor which is chiefly distal but may affect any joint of the limbs. There is slight tremor in movement, also, but no ataxia. Sensation is unaffected. All the deep reflexes are exaggerated, but there is no clonus. The plantar reflexes are both flexor.

The patient has mitral stenosis, systolic and presystolic murmurs being present at the apex. Her visceral organs are otherwise normal.

The cerebrospinal fluid is normal and the Wassermann reaction in her blood is negative.

Diagnosis.—The condition is regarded as Parkinsonism, most probably subsequent to an attack of encephalitis lethargica. The possibility that the original lesion was due to a basal embolism is improbable in view of the later gradual progress of the symptoms. The chief interest of the case is that the right side of her face and her left limbs only are now affected by tremor. This distribution of the symptoms is most probably due to the chance distribution of a bilateral lesion.

Section of Laryngology and Section of Otology.

COMBINED SUMMER MEETING, JUNE, 9, 10, 11, 1927.

OTOLOGICAL SESSION, JUNE 10, 1927.

DISCUSSION ON PROGRESSIVE MIDDLE-EAR DEAFNESS.

A Preliminary Study of Chronic Middle-ear Deafness.

By GEORGE SCOTT WILLIAMSON, M.D., and
E. HORACE RICHARDS, F.R.C.S.Ed.

(I) GEORGE SCOTT WILLIAMSON, M.D.

A PRELIMINARY analysis of the data acquired from the study of cases of chronic deafness, seems, I think to indicate the lines along which the investigation into its ætiology might be pursued.

(1) Cases were selected and classified so as to include only idiopathic deafness. From this group congenital deafness was also excluded.

The questions presented to us were: "Do these cases represent a clinical entity?" and secondly, "Do these cases represent deafness as a sign of any constitutional condition or disease?"

Procedure.—Cases were admitted to the hospital on Friday afternoon and watched until Monday morning; in some cases on two occasions. They were put on a fixed diet of more or less known value.

At the first three sittings a history was obtained, and the parents and friends interviewed. A full and careful examination was made of each system—cardiac, respiratory, digestive, urinary, neuro-muscular, genital and hæmopoietic.

The urine and blood were taken on the Saturday morning, and the Sunday evening for comparison, and submitted to a full chemical examination. A sugar-tolerance test was carried out; also a lævulose test for hepatic function, a urea concentration test for renal function, a test for the acid-alkali balance in the tissues, and for the vital capacity (lung). The blood-pressure and the cardio-respiratory balance were estimated.

It is not contended that these tests add materially to the information derived from the ordinary clinical examination so far as the detection of disease is concerned. I was interested in them then for a purely scientific reason of a more general nature. They may, however, serve to confirm clinical findings.

General Results.—Fifty-one cases were examined; seven presented signs of established disease, namely, mitral disease of the heart (three cases); Graves' disease (three cases, one in a male); tuberculosis (one case).

The patients in these cases were submitted to treatment and improved materially in their general condition, with the result that the tinnitus was considerably diminished and ceased to be a source of *continuous* complaint; indeed I have taught these seven patients to use their tinnitus as an index for treatment.

Special tests (excluding the seven cases of disease)—With one exception (the cardio-respiratory balance) the result of all the special tests carried out indicates that in these forty-four cases the patients were within the normal of average people at their age limits and need no further discussion. They all showed a decided tendency to tachycardia, and had an easily disturbed cardio-respiratory balance, such as one sees in fatigue, asthenia, etc.

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Negative results especially noted:—

(1) No emphatic history of nasal catarrh.
(2) No history of tonsillitis or adenoids.
(3) No marked susceptibility to colds.
(4) Teeth crowded but no marked dental caries.
(5) No constipation.
(6) No low weight levels.
(7) No general body deformity.
(8) No defective eyesight.
(9) No mouth breathing.

Positive signs noted:—

(1) *Age.*—Forty out of fifty-one cases occurred between the ages of 18 and 30. The greatest incidence is at 22 years.

This does not, however, represent the actual state of affairs. The disease is a chronic one; its duration is from three to seven years as noted by the patients. All had a vague history up to the age of 10 years, and the older the patient the older the history. The duration of the disease as stated by the patient is that of the tinnitus rather than that of the deafness. As already indicated, the tinnitus is a fleeting symptom even in old-established disease. We may therefore conclude that the deafness preceded the tinnitus. So that ten years' duration is the minimum possible.

Now, if we take the incidence as twenty-two years and subtract from it the average of ten years' minimum duration, that brings us to the age of puberty This is the latest—not the earliest—age at which the deafness can begin.

The disease, therefore, must begin in school life. A fact supporting this probability is that in all the cases shown there is a marked degree of dullness, apathy, bad speaking, etc. I suggest that this is due to an actual deafness, or at least to a low auditory acuity during school years.

Auditory acuity is naturally of greater importance than deafness itself, a confused hearing is quite as disastrous in education as confused vision. School authorities lay far greater stress on auditory acuity than on visual acuity, but as yet we have no means of estimating auditory acuity—a rather serious reflection on the specialty!

(2) *Facies.*—Another positive fact noted was that in each of the fifty-one cases the patient had a peculiar aspect, depending upon the size and shape of the nose. The nose was small and thin, with a delicate bridge and attenuated skin; the alar muscles were atrophied and the nostrils were mere slits.

The patients were not mouth breathers. Mouth-breathing was tested for, by the method I use in studying adenoid children. The mouth is closed with a gag and the pulse and respiration are watched—in a few cases a blood analysis was also done. These patients had no difficulty in breathing through the nose to the necessary respirator capacity.

I would suggest that this facies should be studied—it suggests atrophy of the nasal organ. I regret I did not include a test for smell as part of the inquiry. Perhaps a special X-ray study of the bones may be of value.

(3) *Tachycardia.*—The third positive sign (already mentioned), and a decided nervous instability, are part of the "make-up" of these cases.

Distribution between the Sexes.

There were five males in fifty-one cases. Hospital statistics cannot decide whether this proportion represents the actual distribution between the sexes. The disease is a chronic one and it begins in childhood. The male, being under greater compulsion to adapt himself, probably ignores his deafness, or has no leisure for hospital attendance, except with regard to "compensation" disabilities. Further, the male youth has a choice of occupation, and will, at the outset, choose work not depending on auditory acuity. Four of the five males worked in noisy repetitive occupations—engineering, road-mending, etc. Women have leisure and a limited choice of occupation.

Thus the fifty-one cases may represent a differential selection of cases rather than an actual incidence of the disease. Statistics from private practice might clear up this point, since the reasons just stated have less weight with this class of patient.

Conclusion.

Deafness with nervous tachycardia and a deformity of the nose (pinchbeck) has its onset in childhood. It is not associated with any disease, but may be associated with some constitutions or diathesis. It is not due to local sepsis.

A test for auditory acuity as distinct from deafness is urgently needed.

This note seems to indicate that this deafness is not merely an accident during the course of life. It has a specific ætiology somewhere in childhood so that further study would be well worth while.

(II) E. Horace Richards, F.R.C.S.Ed.

Analysis of Hearing.

It was decided last year to investigate at the Central London Throat, Nose and Ear Hospital, a series of patients suffering from chronic deafness of the type commonly known as "chronic middle ear catarrh," an ill-defined state variously called by authors, otitis media catarrhalis chronica or sicca, otitis media sclerotica, hyperplastica or adhesiva lentescens (Politzer), and dry, adhesive or atrophic catarrh (Dan McKenzie), or chronic hyperplastic otitis media (Kerrison); all these terms emphasize a slowly progressive fibrotic process, of an inflammatory or catarrhal nature, occurring in the middle-ear cavity.

The object of this investigation was to seek some indication of the lines along which research into the real cause of this deafness might be most profitably pursued.

Cases were selected from among the out-patients of the hospital by the surgeon on duty, and referred to the in-patient department for a further investigation, which consisted of a general physical and pathological examination, and a routine examination of the nose, throat and ears, including only the simpler and generally accepted auditory tests; the conduct of the latter was entrusted to myself. The result of this local examination, unfortunately limited to ten cases, will form the basis of this short statement of observed facts.

The data may conveniently be considered under three main headings:—

(1) *Symptoms.*—(*a*) Complained of by the patient; (*b*) facts elicited by interrogation.

(2) *Findings* on examination of the nose and throat, with special relation to the evidence of local inflammation, and of the ears.

(3) Response to *hearing tests.*

All complained of deafness, the duration of which varied from two to ten years or longer, but the onset in all but one case (No. 2) was indefinite as to time, and progressive increase was the invariable rule, one ear being affected earlier and more severely than the other. The one exception noted was a case of sudden onset of deafness in the left ear following influenza four years ago, but the patient (a female) did not admit that there was any deafness in the other ear, although testing revealed definite impairment. This case demonstrates that the patient's history may be unreliable as to the time of onset of deafness.

All except one patient (No. 10) complained of tinnitus, but they were even more indefinite as to its duration. Only three patients could give any estimate at all; the patient in the case of sudden deafness said the tinnitus had lasted four years, the deafness and tinnitus apparently coming on together; another said it had

been present five years (he having been deaf for at least ten years); while the third said she had always had a pulsatile tinnitus, rhythmical with the respiration; in fact, previously to seeking medical advice for the deafness she thought it "natural for everyone" to have this symptom.

The sounds heard were mostly single, variously described as of escaping steam, "shell sounds," humming, buzzing, roaring, rumbling, the rushing of water, the noise of machinery, and of bells—the "news bells." In four cases, combinations of sounds were heard, up to four simultaneous components being appreciated. In no case was there any suggestion of ideation.

In response to interrogation, several other symptoms were brought to light:—

(1) *Vertigo*, occasional giddiness, "when run down"	2 cases	
„ „ on turning rapidly	1 case	
Definite attacks of rotatory vertigo, with tendency to fall, and increased tinnitus	2 cases	
Vertigo and falling after Valsalva inflation	1 case	
(2) *Paracusis*	3 cases	
„ doubtful	2 „	
(3) *Nasal*, recurring "colds"	2 „	
„ "catarrh," nasal 2	8 „	
„ „ post-nasal 6		
(4) *Laryngeal*, recurrent huskiness	5 „	
(5) *Pain*, earache	1 case	
„ „ history of same in childhood	1 „	
„ headaches	2 cases	
„ referred to neck	1 case	
„ „ throat	2 cases	

A history of deafness in the family was obtained in one case only.

On routine examination, there were found to be: men, three, women, seven, of ages varying from 20 to 51 years, with the maximum age-incidence between 21 and 28 years, somewhat higher figures than are given by a consideration of the whole series.

Of the nine cases with nasal or pharyngeal symptoms, evidence of any active inflammation could be found in three cases only:—(*a*) Small pads of inflamed adenoids and granular pharyngitis (two cases); (*b*) redness of nasopharynx with mucus around Eustachian cushions (one case).

In the other cases, varying degrees of septal deflection, without any evidence of infection, could be found.

All cases were transilluminated, and showed good illumination of the maxillary antra and other regions.

The larynx in all cases appeared healthy.

Marked dental disease was conspicuously absent. In two cases alone there were a few somewhat carious teeth.

On the basis of this examination, the series of cases was divided into two groups:—(1) With healthy nose, nasopharynx and pharynx. (2) With naso-pharyngitis; and it will be shown later that these two groups are further marked by certain characters of hearing.

Ears.—In all cases, externally normal, cerumen was noted to be present only twice, and then in small quantity.

The tympanic membrane was noted to be quite normal in one case only. The typical changes found include the following:

(1) Injection of vessels, especially in relation to the periphery of the postero-superior quadrant and the handle of malleus. This phenomenon, noted in four ears, was in each case accompanied by deficient air-entry on Eustachian catheterization, inflation producing improvement in hearing in one case only of these, and in one other case with widely-patent tubes, but with lax and thinned membrane.

(2)	Loss of normal lustre	18 ears
(3)	Indistinctness, deformity or loss of "light reflex"	16 „
(4)	Mottling of surface	2 „
(5)	Opacity, localized	2 „
	„ generalized	1 „
(6)	Thinning, localized to superior part	5 „
	„ generalized, with in-drawing	2 „
(7)	Apparent fixation of malleus	3 „

Slight spontaneous nystagmus was noted in one case.

Caloric reaction gave diminished response with both ears in one case of severe deafness, a history suggestive of interstitial keratitis in childhood (No. 6).

In considering the response to hearing tests, the cases will be divided into two groups:—(1) Cases with no discoverable nasopharyngeal inflammation, and (2) including the three cases previously mentioned.

Range of hearing for	Groups I and II	Group I	Group II
Soft Conversational Voice.—			
15 ft. and over	8 ears ...	2 ears ...	6 ears
0 to 4 ft.	12 „ ...	12 „ ...	0 „
Soft Whispered Voice.—			
15 ft. and over	3 „ ...	0 „ ...	3 „
3 ft. to 7 ft.	4 „ ...	1 „ ...	3 „
0 to 2 ft.	13 „ ...	13 „ ...	0 „
Watch Tick, normally heard 6 ft.—			
18 in. and over	3 „ ...	0 „ ...	3 „
0 to 8 in.	17 „ ...	14 „ ...	3 „
Lower Tone-limit.—			
32 D.V.p.s.	4 „ ...	0 „ ...	4 „
64 „	6 „ ...	4 „ ...	2 „
128 to 256 D.V.p.s. ...	10 „ ...	10 „ ...	0 „
Higher Tone-limit.—In all cases lower than normal, difference indicated by figure on scale—			
Difference of 0 to 15 ...	10 ears ...	4 ears ...	6 ears
„ 16 to 27 ...	6 „ ...	6 „ ...	0 „
Doubtful, probably over 15	4 „ ...	4 „ ...	0 „
Rinne Test.—Fork = 256 D.V.p.s.			
Positive response ...	3 „ ...	0 „ ...	3 „
Negative „	15 „ ...	12 „ ...	3 „
Borderline „	2 „ ...	2 „ ...	0 „
Fork = 512 D.V. p.s.			
Positive response ...	0 „ ...	0 „ ...	0 „
Negative „	11 „ ...	11 „ ...	11 „
Borderline „	5 „ ...	1 „ ...	4 „
Undetermined	4 „ ...	2 „ ...	2 „

Gelle's Test.—

Positive response	...	11 ears	...	5 ears	...	6 ears
Negative „ (fixation)	7 „	...	7 „	...	0 „
Reversed positive response	2 „	...	2 „	...	0 „

An examination of the above tabulated figures shows (1) That the characteristics of the whole group are those of deafness of the "mixed" type; (2) that in the few cases comprised in the two groups there is a qualitative difference only.

Those in Group 1, the more numerous, have, with only one exception, a very high degree of deafness, in fact, a diminution in acuity of from 90 per cent. to 95 per cent. combined with a peripheral contraction of the tone limits, with the usual accompanying alterations in the relation between acuity of hearing by bone and air conduction.

The Gelle and Weber tests are notoriously unreliable and difficult of interpretation, and the results obtained in this series hardly justify their inclusion.

Mr. G. J. Jenkins.

When I was asked to take part in this discussion I replied that I had not been able to recognize, pathologically or clinically, the condition known as "progressive middle-ear deafness," sometimes described as "chronic middle-ear deafness," or even termed "dry middle-ear catarrh." The answer was: "Say *that*"! I have not heard these terms applied to any form of middle-ear disease in the aural department under the care of Mr. Arthur Cheatle at King's College Hospital, and from my own observations have come to the conclusion that the terminology is wrong and misleading.

We are all familiar with the text-book description of the disease under this name, the detailed accounts of the active local treatment and the general management of the patient. The pathology is in most books obviously speculative. The symptoms and signs are usually roughly and indefinitely indicated, and so far as they go, are the same as an imperfectly described otosclerosis syndrome. I believe that if a complete modern examination had been made in these cases on which this terminology is founded, the diagnosis would have been one of otosclerosis.

In my opinion, pure middle-ear changes, including fixation of the stapes, will give only pure obstruction signs, and if any symptoms such as internal ear deafness, or those especially associated with otosclerosis, are present, we should then regard the labyrinth as affected. I have not known middle-ear deafness which could be attributed to middle-ear changes only to be progressive in the sense implied in the title of this discussion.

From the foregoing statements it will be deduced that I have very little to say about treatment, but if there had not been that persistent description of this class of case in text-books, many patients would have been saved the effects of disappointed hopes.

Dr. Dan McKenzie.

What is Progressive Middle-ear Deafness?

We have heard much in this Section about one of the varieties of progressive middle-ear deafness, namely otosclerosis, chiefly with reference to the mystery of its ætiology, but the other varieties have, I fear, been somewhat neglected; until now, it may be by reason of this very silence, there are those who doubt whether any other varieties exist at all, and suggest that progressive middle-ear deafness is merely another name for "otosclerosis."

That being so, a clinical review of some of the cases I have seen personally may be of interest.

In the following survey I have confined myself to my private cases, simply because they had been under the control of one observer.

In all, 748 cases are reviewed. No selection has been made except that those only are dealt with in which the deafness had advanced beyond the stage of mildness, that is to say, in which the hearing for the voice was less than 10 ft., and for the middle C tuning-fork at least 15 sec. less than normal. I wished, in other words, to deal with a definite clinical picture.

As to diagnosis and classification, I have as far as possible followed the text-book division into: (1) Chronic dry catarrh of the middle-ear; (2) cicatricial changes secondary to healed suppuration, what we sometimes call "residual suppuration"; (3) active suppuration; and (4) otosclerosis.

In effecting a distinction between catarrhal and otosclerotic deafness, reliance was placed upon the presence or absence of "colds" in the history, and upon the presence or absence of benefit from inflation.

Admittedly, however, these criteria are not perfectly reliable, and so many of the cases diagnosed as catarrhal ought to have been placed in the otosclerotic group. On the other hand, it is at least conceivable that the reverse holds good. Here, then, at the outset of our investigation we encounter a source of fallacy which must to some extent vitiate our conclusions; but this is inevitable.

In the "suppurative" group, only those cases are included in which the history shows the deafness to have been progressive. Stationary or arrested cases are excluded.

Many cases of progressive deafness could not be classified at all. They also are therefore excluded.

As to *numbers*: catarrh provided 288; residual cicatrization, eighty; active suppuration, 136; otosclerosis, 206; while thirty-eight were, or seemed to be, a combination of suppuration, healed or active, with catarrh or otosclerosis.

Sex Incidence.

A statistical calculation for catarrh, made in response to a request by Dr. Scott Williamson, and based upon a larger number of cases than those adopted for my present purpose, gave 340 females and 199 males, or roughly, 3 : 2.

Residual Suppuration.—In the eighty cases mentioned above, fifty-four were females and twenty-six males.

Active suppuration of one or both ears (136 cases), sixty-nine females, sixty-seven males.

Otosclerosis (206 cases), 145 females, sixty-one males, roughly 5 : 2.

Summing together the figures for sex incidence, we find 608 females and 353 males, showing a definite preponderance of females: this preponderance is greatest in the otosclerotic group.

We may note that in forty catarrhal cases, or 14 per cent., and nineteen otosclerotics, or 9 per cent., the deafness was unilateral. My impression, however, is that evidence of pathological change exists almost invariably in both ears, but that some slight difference in its distribution has led to the escape of one ear from deafness. This impression almost becomes a conviction when one hears how frequently such an escape is only temporary.

Age of Onset of the First Symptom.

Influenced by Dr. Scott Williamson's arguments, I have gathered together all the available data bearing upon this detail. My figures do not, as will be seen, altogether coincide with his.

In most cases, certainly, tinnitus is the earliest symptom. Generally speaking, it precedes the deafness by months and sometimes even by years, and it is a common

experience that persistent tinnitus is followed by deafness in almost all cases. Sometimes the tinnitus and deafness appeared simultaneously. In only one catarrhal and four otosclerotic cases was there no tinnitus at all, although in many it is noted as being only mild or occasional. Tinnitus of the composite type, that is to say in which several different subjective sounds are heard simultaneously, was found to be frequent in otosclerosis. Indeed, of the classical high-pitched tinnitus described as typical of otosclerosis I have not recorded a single instance. Composite tinnitus was also complained of in many of the cases diagnosed as catarrh. The onset of persistent tinnitus in suppurative cases, whether residual or active, may generally be taken as an indication that the deafness is becoming progressive.

It is frequently impossible to obtain from a patient the date of onset of the malady, so that the numbers here given are less than the total number of cases examined. Probably most patients assign too short a duration to their malady. But as this tendency applies to all cases it does not vitiate the *relative* value of the figures.

In catarrh, the age of onset was given in 150 cases. One referred it to "infancy" and eight to "childhood." The greatest age given was 72, and in two cases the age was 65.

The average age of onset in this group, counting "infancy" as 3 years, and "childhood" as 6, was 32 years.

In otosclerosis the age was given in 122 cases. Nine were referred to "childhood," and one to 10 years of age. The oldest was 60. The average age at onset in otosclerosis was 26.

Tuning-Fork Tests.—It is generally taught that progressive middle-ear deafness is of the obstructive type, the bone-conduction for middle-toned forks being increased. The results I have obtained, however, indicate that this teaching should be modified.

The comparisons made during the twenty or more years covered by these observations have been estimated by the use of no fewer than six different persons, all in my own presence, and without any decided discrepancy in the results. Nevertheless, my figures may possibly be inaccurate. If so, I hope that some other observers will undertake a similar statistical inquiry based upon their own experiences, as the point at issue is one of some importance.

The following are the results obtained, using tuning-forks, 256 V.D.

(1) Of 431 catarrhal ears tested and noted, 291 showed increased bone-conduction (+ B.C.); 80 showed neither increase nor decrease (± B.C.); and 160 showed diminished bone-conduction (– B.C.).

(2) Of 154 ears with residual suppuration (cicatrices and perforations) 88 showed + B.C.; 16, ± B.C.; and 50, — B.C.

(3) Of 139 ears with active suppuration (excluding labyrinth cases), 95 showed + B.C.; 12, ± B.C.; and 32, — B.C.

(4) Of 363 otosclerotic ears, 224 showed + B.C.; 53, ± B.C.; and 86, — B.C.

I ought perhaps to say that a + B.C. in one deaf ear with a — B.C. in the other, also deaf, was quite a usual experience.

If we take the ± and — cases together as representing a diminution in the normal bone-conduction of obstructive deafness, we obtain the following proportions :—

	Bone-conduction +	Bone-conduction —
(1) Catarrhal	5	4
(2) Residual suppuration ...	4	3
(3) Active suppuration ...	5	2
(4) Otosclerosis	5	3

(In this estimation we must, of course, have omitted many otosclerotic cases in which the only signs present were those of pure nerve deafness.)

This table, and the foregoing statistics generally, if reliable, are interesting for three reasons: (1) They support the belief that the text-book groups do really represent, however roughly, a natural division. (2) They indicate that the highest proportion of — B.C. cases occurs in the catarrhal and residual suppuration groups. [Is this an effect of scar tissue?] (3) They show that the traditional teaching requires modification, for if we sum up all of these figures and average them, we find that three out of eight cases of progressive middle-ear deafness show a diminished bone-conduction.

A diminished bone-conduction was often encountered even in early cases with only trifling deafness, and its presence seems to be of no value in prognosis. It does not necessarily signify that the deafness will advance more rapidly or become more severe than when the bone-conduction is increased. To this opinion there may perhaps be some opposition.

Although, however, the bone-conduction was found to be so frequently diminished, the amount of diminution, measured in seconds, is slight; much less, relatively to the deafness for air-borne sound than the diminution registered in what we diagnose as pure nerve-deafness. For example, with deafness equal to — 20 in. at the meatus, the bone-conduction would only be — 5 in. Further, one point in the traditional teaching is invariable, namely, the Rinne in all was negative.

Low Tones.—In all the groups the bone-conduction for the low forks also frequently showed a diminution. But it is only within recent years that I have begun to test the bone-conduction for low forks, and so I do not give any numbers. Lately, however, at the suggestion of Mr. G. J. Jenkins, I have been testing it in otosclerosis, and have found diminution in fifteen, and increase only in two, out of seventeen cases. Thus it would seem that the bone-conduction for the middle forks in otosclerosis is generally increased (see above Table, as 5 is to 3), while for the deep forks (64 D.V.) it is generally diminished. This diminution, however, is not a certain indication of otosclerosis, as I have recently found it present in what seemed to be simple Eustachian catarrh.

My figures support the orthodox teaching that in progressive middle-ear deafness the hearing for deep tones through the air is reduced relatively to the hearing for middle and moderately high tones.

The upper tone limit was tested sometimes with Galton's whistle, sometimes with the monochord, sometimes with both. The following changes were recorded:— (1) The upper tone limit was reduced, allowance being made for age, in seventy-four catarrhal ears; in six with residual suppuration; in three with active suppuration; and in eighty with otosclerosis. Thus the otosclerotic group showed the highest proportion. (2) We have just drawn attention to a discrepancy between the bone-conduction for the middle tones and that for the lower tones. Here also a discrepancy was found in another direction, namely, between the bone-conduction for the middle forks and the air-conduction for the higher tones. It was as follows: The upper tone limit was lowered, although the bone-conduction was increased, in thirty-four catarrhal ears, in two with residual cicatrices, in one with active suppuration, and in thirty-eight with otosclerosis. Once again the highest number occurs in the otosclerotic group.

But, as with the diminished bone-conduction, so with the upper tone limit; although in these cases it was lowered, it did not reach so long a reading as in pure nerve deafness, averaging about two in the Galton, or about 17 cm. in the monochord.

Physical Examination.

I have here to record a personal finding which again may arouse criticism, namely, that the condition of the membrana tympani—its colour, texture and

situation, together with the mobility or immobility of the malleus handle, as tested by Siegle's speculum—is no indication of the presence or absence of deafness, still less of its degree. I have frequently found the malleus handle immobile, adherent indeed to the promontory, in both ears, one with normal hearing and the other with only trifling deafness (− 6 in. A.C. and − 3 in. B.C. middle fork, in one case). On the other hand I have found the malleus handle moving, to all appearance normally, in the one ear and adherent and fixed in the other, and yet the ear with the movable malleus was deafer than the ear with the fixed malleus.

I began by collecting records of these findings also for statistical purposes, but soon gave up doing so, as they seemed to be of no value.

In the same way, and for similar reasons, I abandoned summarizing the condition of the pharyngeal orifice of the Eustachian tube as seen through the endoscope. In otosclerosis, certainly, it is, as a rule, quite normal. But in catarrhal cases it may be found in any state—normal, gaping, collapsed, or swollen, and the respective numbers are about equal.

(It is perhaps worth recording that out of the 206 otosclerotic cases, six were found with hyperostosis or exostosis of the bony wall of the external auditory meatus.)

If, now, we detach our minds from our classification into four groups, and regard those cases as a whole, we find that they manifest five points in common, though varying somewhat in their incidence: (1) Tinnitus, frequently composite; (2) progressive deafness; (3) bone-conduction variable, but increased for the most part; (4) upper tone limit also variable; and (5) a negative Rinne.

The fact that this symptom-group is common to all the varieties, surely indicates that, whatever may be the primary pathological change that initiates the process, there must be some physical factor, also common to all the groups and varieties, that induces the syndrome. It would also seem that once this physical factor—this secondary change—whatever it may be, is set going, it will continue to progress by its own inherent tendency, and independently of its initial cause, until, again of its own inherent tendency its progress comes to a standstill. We all know, for example, how unsuccessful are all our efforts to cure, or even to arrest, this type of deafness by the cure of catarrh which originated it. Yet it may also be said that the disease-process is a self-limiting one. It does not progress to the entire destruction of function.

What may be the nature and situation of this physical factor does not seem to be quite obvious even at the present day.

Sometimes the fault is attributed to the immobilization of the membrane and of the ossicular chain as a whole. We cannot deny that this element, when present, may definitely reduce the hearing-power, since it interposes an impediment to the transmission of sound-waves to the cochlea. Moreover, the gradual progress of the deafness can be explained by the gradual stiffening of the several ossicular articulations. On the other hand, we cannot help wondering whether the membrane and ossicles have much to do with sound-transmission, when we come across cases with almost perfect hearing after the radical mastoid operation. Again, the observation above noted, of the combination of an immobilized malleus with normal hearing, is difficult to explain on this assumption. For these and other reasons, the reference of the ossicular impairment more particularly to the foot-plate of the stapes, is surely a view more worthy of our support.

We have also to consider the effect upon the intracochlear sound-transmission produced through immobilization of the membrane of the round window by scar-tissue. These reflections, it will be seen, guide us to assuming the inner wall of the tympanum to be the seat of the physical factor for which we are searching.

But we have still to account for two phenomena which recall to mind the nerve-mechanism of the cochlea: (1) the tinnitus, and (2) the not infrequent presence of a diminished bone-conduction.

Tinnitus is undoubtedly produced in the cochlea, at least in the early stages. Later on, as Lake's experiences in operating on the labyrinth have clearly shown, the morbid sound is central in origin, for the destruction of the cochlea generally fails to silence it. I think we may say that tinnitus is due to irritation of the cochlea, but that is about as far as we can go. It is noteworthy that the tinnitus of acute otitis media is, as a rule, different in quality from the tinnitus of progressive deafness, in being a rushing and often a pulsating sound. Obviously, the former variety is vascular in origin, and perhaps it is due less to congestion of the middle-ear than to a sympathetic hyperæmia of the adjoining cochlea.

But the tinnitus of the disease we are now considering is not due to active congestion, since the pathological lesion is either a cicatricial or an osseous change from which hyperæmia is apparently absent. Can it be due to increased intra-labyrinthine fluid pressure?

As to the diminished bone-conduction and the lowered upper-tone limit, these also suggest cochlear changes. Yet, as I have shown on a former occasion, these phenomena may be due to simple obstruction since they may disappear when the obstruction is removed. I have even observed them in deafness from impacted cerumen.

That being so, we may, I think, refer even such apparently cochlear phenomena to the middle-ear, and, if, as seems plausible, we may credit the fixation of the fenestral structures—whether by bone as in otosclerosis, or by scar-tissue as in suppuration and the dry catarrhs—with the power of causing tinnitus by altering intra-labyrinthine fluid-pressure in some way, we are in the position of being able to refer all the phenomena to the middle-ear, and particularly to the inner wall.

This argument, let me be careful to add, does not exclude the real occurrence of genuine cochlear changes and cochlear deafness as a result of the permanent withdrawal of normal cochlear stimuli attendant upon serious middle-ear deafness—changes which we know do undoubtedly occur.

Mr. H. Norman Barnett.

In my opinion this condition may be divided into two classes: (1) Those following long-antecedent middle-ear suppuration, or those secondary to infection from septic tonsils and to obstructed airway caused by deflection of the nasal septum; (2) those arising without these conditions being present, but almost certainly due to infection through the Eustachian tube from some other cause.

In the case of those following old middle-ear suppuration, the condition of things, then present, leads to uncured deposits being left behind. In the case of the other types, the same thing from a different source has occurred, and the material gradually forms adhesions between the ossicles and sometimes between the ossicle and the tympanic membrane. As time proceeds this material becomes more organized and the ossicles become more and more incapable of movement till finally no oscillation takes place. This process is usually very gradual and the result is that the onset of this type of deafness may occur long before the patient is seen and the condition is spread over a large number of years. The condition thus produced results in the sound waves being unable to traverse the tympanum and thus we get complete or almost total deafness although there is no lack of conduction by the nerve and no otosclerosis is present, though in some cases tinnitus is a marked symptom.

The treatment I carry out in these cases is designed with a view to causing absorption of the deposited material and breaking down the adhesions, by means of oto-massage of the tympanic membrane, ionization over the mastoid or in the meatal passage—preferably the former—and the passage of iodine, camphor and carbolic acid vapour through the Eustachian catheter to the middle ear. The time of this treatment will vary from three weeks to three months, according to the nature of the case and the state and organization of the deposit.

82 Barnett—Fraser: *Progressive Middle-ear Deafness*

The three cases[1] to be shown to-morrow are illustrative of: (1) A cure which may be regarded as complete for all practical purposes—the patient has not been seen for two years, but he is able to perform his professional duties with ease; (2) a case now under treatment and rapidly improving so that this patient also is now able to do his work without hindrance; (3) a case also under treatment but not so far advanced and of a slower type in responding to treatment. In each of these cases there had been a preliminary operation on nose and throat, this being indicated on general medical principles.

In my opinion many cases of deafness are "turned down" and the statement made that nothing can be done, because the value of the above recorded type of treatment is not recognized and in some cases it is thought that otosclerosis is present. I believe otosclerosis to be a comparatively rare condition; many so-called cases are really those of chronic or very advanced types of progressive middle-ear deafness.

Combined with the above treatment a most important factor is the re-education of the hearing centre, for after all it must be remembered that we—so to speak—hear with our brain rather than with our ear, which is a conducting and selecting apparatus. In the deaf, especially those who have been so for a long period, the centre becomes dormant owing to the fact that sounds have not been reaching it; and even when, after treatment has been begun, the sounds begin again to travel through the middle ear and are conducted by the auditory nerve to the centre, there is little or no response and the patients have to be warned to respond to any stimulus of sound that they hear, whether they are wrong in its interpretation or not. Some persons, according to their temperament, are much more difficult to re-educate than others. The best re-educative medium is the patient's own voice, conveyed to the centre through a long binaural speaking tube.

Dr. J. S. Fraser

said that in spite of the papers which had been contributed, the subject was still enveloped in a fog. He found considerable difficulty in knowing what kind of case Dr. Scott Williamson had discussed. Dr. Williamson spoke of "idiopathic" deafness—a term often employed when little was known about a subject—and added that he had excluded cases of congenital deafness. If Dr. Williamson referred to otosclerosis, the proportion of cases in which the deafness was inherited was over 50 per cent. Therefore he must have excluded many cases of otosclerosis from his statistics, and yet he spoke about the facies of the patients, etc. It was in regard to otosclerosis that some had said—he thought with truth—that one could almost "spot" the case when the patient entered the room. It would have been better if Dr. Williamson had had his cases classified by an otologist before he examined them.

He (Dr. Fraser) thought that the best classification of these cases of chronic deafness was the following: (1) Those with a normal, and (2) those with an abnormal, tympanic membrane. If one had to deal with a chronic deafness in which the patient had a normal drumhead and yet gave the usual tuning-fork results of obstructive deafness, one could be sure it was otosclerosis, whether or not there was a flamingo tinge in the promontory. If that tinge was present it only clinched the diagnosis. Further, he wondered whether a considerable number of cases which gave the tuning-fork tests of nerve deafness, in young or middle-aged people, were not really instances of otosclerosis. Shambaugh, of Chicago, pointed out that many cases of nerve-deafness really had "atypical otosclerosis." These patients had a well marked family history of deafness but, on functional examination, one found that bone conduction was diminished and Rinne's test was positive. Shambaugh stated that some of these cases had been confirmed by microscopic examination after death. Shambaugh's view was that in these cases the "osteitis vasculosa" affected part of the cochlear capsule but did not involve the region of the oval window. If the latter were affected, one got the test results which one expected in otosclerosis.

He (Dr. Fraser) agreed with Mr. Jenkins that in most cases with well marked abnormality of the drumheads the deafness was not progressive. These people had suffered from otitis media in childhood and otoscopy showed the presence of retracted scars in the drumhead, chalk patches, etc. Yet these patients, as a rule, heard very well until they reached the age when the changes characteristic of senile nerve deafness might be expected. When there was a really abnormal drumhead the recognition of otosclerosis was more difficult than when the tympanic membrane was normal, but if tinnitus was a marked feature in the case, if

[1] See p. 102.

there was a hereditary history of deafness, if the catheter gave no improvement in hearing and if the deafness was progressive, one was justified in regarding the case as one of secondary otosclerosis.

He (the speaker) was surprised at the President's statistics, which gave a high proportion of chronic catarrhal otitis media in comparison with otosclerosis. In most of the books now being published, chronic catarrhal otitis media was receding into the background while otosclerosis was markedly advancing. His own view as to the proportion of the two affections would be very different from that shown by the President's figures.

Sir JAMES DUNDAS-GRANT

said he could not produce the material which he thought ought to be brought before the Section, namely, post-mortem specimens to illustrate the actual condition found in these cases. He still looked to some of the younger workers in the specialty to produce this kind of material, which was much wanted.

Many large and small questions arose for discussion. For instance, what was the influence of pure obstruction of the Eustachian tube? His (the speaker's) view was that many cases of chronic progressive middle-ear deafness were secondary to the narrowing of the walls of the Eustachian tube. He was inspired by a case shown by Mr. Tilley and by one he had seen himself, to quote a paper of Krainz's on that subject.[1] The case recorded was one in which, from compression of the Eustachian tube by a sarcoma in the nasopharynx, an exudative serous catarrh and thickening of the lining membrane of the mastoid cells had followed, apparently as the result of the mechanical obstruction. Every effort should therefore be made to improve the condition and patency of the Eustachian tube.

He did not think pure cases of otosclerosis were common in comparison with those of chronic catarrhal change.

With regard to bone conduction, it must be remembered that a negative Rinne might be due to unilateral nerve deafness, because, when testing bone conduction on one side, it was impossible to exclude stimulation of the nerve on the opposite side.

In many cases of obstruction by cerumen there was not the increased bone conduction which was present in simple Eustachian catarrh—one reason being that there was no increased tension in the middle ear.

Professor Manassez had written an important paper in which he said that many cases regarded as chronic progressive deafness were due to changes in the cochlea. The difficulties met with in analysing these were sometimes enormous.

Mr. SOMERVILLE HASTINGS

said he would briefly discuss the question of auditory intelligence. Some years ago Major Tucker and himself had brought before the Section an apparatus for testing patients' hearing power—an audiometer. He had used it for several years but had not brought his results before the Section, as they were so indefinite; the question of auditory intelligence entered so much into the problem. He agreed that there was need of research on this question.

He also was struck by the proportions given by the President between the chronic catarrhal and otosclerotic types of chronic deafness. He (the speaker) was a heretic in this matter, as, in his experience, otosclerosis was very rare. He could only remember two cases in which, with the ordinary signs of middle-ear deafness, the tympanic membrane, the nose and the nasopharynx were normal, and nothing suggested a history of infection in those regions. He did not doubt the accuracy of the description of the pathological changes, and admitted that the cases ordinarily called otosclerosis might run in families. But he suggested that they nearly all began with sepsis in some region of the nose and throat.

It was of interest that Dr. Scott Williamson was able to date the onset of these troubles, on the average, from about the time of puberty. His own view was that in many diseases it began in quite early life. Those who examined the ears of young children found that otitis was much commoner than they had suspected, and in a large proportion changes in the ears could be found. It was very difficult to distinguish the cases in which the process persisted, despite all treatment, from those in which it could be arrested. He thought the right course

1 *Journ. Laryng.*, November, 1926.

was to make a very careful examination for a possible source of sepsis, and to treat it when present. It would be found in a sufficient number of cases to render this course well worth while.

Mr. J. F. O'Malley

said it was only natural that there should be differences of opinion among Members on such a subject. If Politzer's text-book, published twenty-five years ago, was consulted on the subject of otosclerosis, a reference would be found to the Bezold triad: (*a*) a normal-looking tympanic membrane, with free Eustachian tube, (*b*) a middle-ear type of deafness, and (*c*) a negative Rinne. Cases of that type were still encountered, and they showed a certain familial tendency, i.e., apparently there was a hereditary factor. Most otologists would probably classify these as otosclerosis.

That there was a definite middle-ear catarrhal trouble which led to deafness must also be conceded, because patients complained of deafness following a cold, and after treatment, when the cold passed away, the hearing was restored. Those were two distinct types. In the former there could also be super-added a middle-ear catarrhal infection. If this did not clear up and an otosclerotic change was progressing simultaneously in the labyrinth, the resultant clinical picture differed from that which either condition separately would present. Shambaugh, in a recent communication, stated that one-third of the patients coming to his clinic on account of deafness had otosclerosis. He (Dr. Shambaugh) thought otosclerosis was now more common than it had hitherto been the custom to consider it, and that was his (Mr. O'Malley's) view also.

He, like Sir James Dundas-Grant, had hoped to hear some encouraging contribution to this subject, and he still trusted that as a result of the discussion help would be forthcoming in elucidation of this difficult problem.

Mr. J. A. Gibb

said that a patient who was practically stone-deaf, except to bone conduction, was willing to have anything done which would improve his hearing. On a hole being made in the postero-inferior quadrant of the drum he was able to hear fairly well. How could that be explained? Judging by appearances, the condition was clearly otosclerosis, yet on perforation of the drum opposite the oval window there was hearing. He had such a patient who now heard as he had not been able to hear for the past eight years. It should be borne in mind that in primary otosclerosis there was an osteoporosis of the round window (an extreme case).

Mr. Broughton Barnes

said that he was surprised to hear Mr. Jenkins deny the existence of progressive catarrhal middle-ear deafness. One could not do anything for the otosclerotic, whereas if the condition was catarrhal one might hope to arrest the process, except possibly for the underlying progressive factor referred to by the President. He did not think Mr. Jenkins would say that a case with thickened drums was one of otosclerosis, and surely those were cases of progressive middle-ear deafness. There were cases in which the drums were greatly thickened, often they were retracted and rather dull. Sometimes in the best ear the postero-superior angle had escaped the thickening, there might be some mobility, and when they were inflated the drums returned to the position in which they were before. Should that be called otosclerosis? A year ago he had had a case which he thought was otosclerosis, the patient, a woman, being aged 23. She had a thin, bright drum, of normal mobility, and he could see a pink glow. She had marked middle-ear deafness and there was a history of family deafness. In this patient there had been three years of progressively increasing deafness. She had a cold, and he gave a nose wash, and on coming back a month later she could hear perfectly. That increased his caution in diagnosing otosclerosis. As an incurable condition, it should only be diagnosed when tympanic causes had been excluded.

Mr. Herbert Tilley

remarked that the President spoke of tinnitus being a cochlear symptom. If that was always so, how did he explain those desperate cases in which suicide was contemplated because of the perpetual noises and for which the auditory nerve had been divided within the skull, and yet the tinnitus continued? He took it that in such cases the tinnitus was of cerebral origin. Did the President contend that a primary cochlear lesion had set up the central change which persisted after the operation for division of the nerve?

Mr. A. R. TWEEDIE

said that, like Dr. Fraser, he could not quite understand the character of the cases described by Dr. Scott Williamson and Mr. Richards. On the one hand, some of them suggested otosclerosis as the patients had heard better when their general health improved—possibly a peculiar characteristic of otosclerosis. On the other hand, fifteen of the cases were definitely associated with a narrow face and ill-developed alæ-nasi, which intimated that here there was a definite intranasal reason for the deafness. He suggested that with a detailed description of the objective examination and results of the functional tests, the value of Dr. Scott Williamson's paper would be much enhanced.

He agreed with Mr. Jenkins' remarks and objected to the term "catarrh"—urging that it would be of assistance both in diagnosis and treatment if this terminology were dropped. If catarrh meant anything, it was only a symptom and could only indicate an excess of the normal secretion. If this description were acceptable, it was obviously quite impossible to talk of "dry catarrh." He did not think there was really any such condition as progressive middle-ear deafness (that is, quite apart from any question of otosclerosis) but he considered that such cases really represented some old past lesion, combined with the normal depreciation of hearing in accordance with years. The latter was accentuated and came into evidence sooner because of such past middle-ear lesion.

He would also emphasize the extreme importance of the functional integrity of the inner tympanic wall, to which allusion had been made by previous speakers—whilst, as regarded the tympanic membrane, so long as it was normally mobile, he did not think that opacity constituted any recognizable factor in loss of hearing, and certainly it was unscientific to describe such opacity as "thickening"—a description which pre-supposed measurement.

He was very glad that this discussion had been introduced, and hoped that it would lead to the establishment of some standard method of examination and standard test, by means of which our observations could be better compared and studied.

Mr. C. HAMBLEN THOMAS

said he was anxious to find out, as a result of this meeting, whether a patient who was progressively growing deaf was becoming so because of catarrh, or whether the deafness was due to some bony change. If there had been some damage to the middle ear through catarrh, an early form of inflammation, it might proceed to cause greater damage later, owing to the fibrous tissue formed. Might not there be such a change in advancing years? And if an ear was damaged, might it not share in any general lowering of the bodily resistance and become more deaf in consequence? Did the otosclerosis mentioned as the cause of increasing deafness by previous speakers, primarily start in the bone, or might any bony changes be secondary to some affection of the mucous lining of the middle ear?

Mr. C. A. S. RIDOUT

said he felt that at the root of all these troubles there was some septic infection of the throat or nose at some time in life. And there was the personal factor: in some patients the deafness progressed, in others it stood still. Once he (the speaker) had had severe suppurative rhinitis, during which his hearing diminished considerably, but his deafness did not progress. He began to suffer from tinnitus thirteen years ago, and he had it still; he did not get colds in the head, and he had no nasal trouble. Some people contracted continuous colds and the deafness progressed—whether they showed signs of otosclerosis or chronic catarrh (terms which could well be abolished). That the condition under discussion should be called by some name all were agreed upon.

Dr. W. S. SYME

said he had been hoping that some light would be thrown on this problem, especially on the question of treatment, but all he heard was that otosclerotic cases should be sent to the radiologist.

He did not think "chronic progressive deafness" was a scientific term, any more than was the term "chronic cough." Cases should be described in terms of pathological changes. He believed there was such an entity as chronic progressive changes in the middle ear.

There was a common type of case in which there was a thickening of the lining membrane of the Eustachian tube and middle ear, probably due to infection by an organism of low

virulence, and the movements which took place caused a re-infection of the parts which was perpetually in progress; this resulted in a chronic adhesive process, which took years to become permanent. Adhesions formed afterwards, and there was a definite condition of the middle ear which constituted what were called chronic middle-ear changes. He did not think such changes put out of consideration the question of otosclerosis as an associated condition, brought about possibly by the nervous tension under which these patients suffered. The right treatment was to discover the origin of the infection. He believed it was always in the nose and nasopharynx.

He did not think very much reliance could be placed on the immediate effects of catheterization in these cases. If there were immediate results from that, so much the better, but he would not regard a case as outside the possibility of improvement because catheterization did not at once make the hearing better. When there were changes in the nose and nasopharynx, they should be treated.

Dr. SCOTT WILLIAMSON (in reply)

said he was not responsible for the nature of the cases which were sent to him. Fifty out of the fifty-one cases presented a picture which made it plain to him that they constituted a type, and he congratulated the clinicians at the Central London Ear Hospital on having clearly defined a condition of progressive middle-ear deafness.

Mr. G. J. JENKINS (in reply)

said that nothing had been said in the discussion to cause him to alter the statement he had made in the opening remarks. He could not recognize a distinct clinical entity "Progressive Middle-Ear Deafness." A middle-ear infection might progress to a cicatricial condition and the clinical symptoms and signs would be the result of this effect only, and could not in his opinion, be progressive in the sense implied in the text-book description of progressive middle-ear deafness.

Otosclerosis was sometimes found concurrent with mild infection of the middle-ear tract, or even with chronic suppurative middle ear disease, but this association did not necessarily imply a causative relationship.

Dr. DAN MCKENZIE (in reply)

said he supposed Mr. Jenkins meant by otosclerosis, bone changes. He (the speaker) did not feel convinced, however, that the change was in the bone. There was a change beyond the catarrhal or suppurative process when a new element was introduced, and then the deafness progressed independently. But he did not think that as yet we were in a position to call that new change otosclerosis, meaning thereby insular osteoporosis of the labyrinth capsule.

The terminology had been criticized, but those in the specialty knew what was meant. The feeling of despair at not doing anything for otosclerosis should not be encouraged. To-day's discussion would have been amply justified if it led to research along effective lines.

A Demonstration of Radiograms of the Normal and Abnormal Labyrinth.

By H. GRAHAM HODGSON, M.B., B.S., D.M.R.E., and F. W. WATKYN-THOMAS, F.R.C.S.

I.—Dr. H. GRAHAM HODGSON.

(Lantern Slides of Radiograms and Diagrams exhibited on Screen.)

I have been endeavouring to obtain satisfactory radiograms of the internal ear, but without much success, until last autumn, when, on the advice of Mr. Jenkins, I went over to Utrecht, where I saw the work being done at the Neurological Clinic by Dr. Stenvers. Dr. Stenvers, to whom I am very much indebted, showed me his method of radiographing the petrous bone, an account of which, as you know, he published some time ago, in the *Acta Oto Laryngologica*. I was much impressed with the radiograms he showed me. As a neurologist, his technique

was chiefly directed to showing the internal auditory meatus, and demonstrating, by means of X-rays, tumours of the cerebello-pontine angle. My interest in the petrous bone was chiefly directed from the otological standpoint, and I hoped that with this in view, by slightly modifying Dr. Stenver's technique, I should be able to work out a series of angles, and demonstrate on a radiogram, the cochlea and canals, and as a step further, to study radiographically the progress of pathological changes occurring in the labyrinth of the living subject.

I will not weary you with details of the technical side of the work. It is sufficient to say that owing to the number, complexity, and density of the various structures overlying the petrous bone, I found it extremely difficult to get what I considered to be an adequate view of the labyrinth. It was only after I had designed special apparatus for doing the work, and had taken 1,200 radiograms of normal labyrinths, that I was able to arrive at the series of angles which I am now using. I am deeply indebted to my colleagues, Mr. Jenkins and Mr. Watkyn-Thomas; they have given me every possible help and encouragement, and taken considerable trouble to look up suitable cases for me to radiograph in the records of King's College Hospital and the Central London Throat Hospital.

I will first show you a series of normal labyrinths. It is at first rather difficult to distinguish the various structures in these radiograms, and it is only after one has looked at a great many that all the structures are immediately obvious.

Radiogram 1 is of a labyrinth, filled with metal, in a dried skull. In it you can see the canals and vestibule, and the cochlea, and internal auditory meatus. To the outer side of the vestibule, and the basal turn of the cochlea, is seen the tympanum, the small shadow in the inner part of the tympanum being the stapes.

Radiogram 2 is that of a normal right labyrinth in the living skull. All the parts shown in this radiogram are very much clearer in the original film, as one necessarily loses a good deal of detail in reproduction on the lantern slide, but I think if you look at the radiogram, and at the accompanying diagram (2a), you will be able to see the various parts of the labyrinth quite clearly. If you follow along the superior border of the petrous bone, from within outwards, you will come to a point in its middle portion where there is seen a light linear shadow running downwards, and slightly inwards, which is the superior canal, the faint shadow crossing its upper part being the subarcuate vein. If you continue in this direction downwards through the vestibule, you come to the first turn of the cochlea, and if you follow this round, you can see the terminal one-and-a-half turns of the cochlea, with their shadows overlapping the shadow of the internal auditory meatus. Just above these turns of the cochlea is seen a small foramen, which is the opening for the seventh nerve. The external canal and lower part of the posterior canal are seen as projections from the outer wall of the vestibule. In this radiogram you will notice the normal arrangement of the capsular bone, which follows the canals closely, and I would also point out the clearly cut margins of the vestibule and cochlea, there being no blurring or haziness of these portions of the labyrinth.

Radiogram 3 is also of a normal labyrinth, in which I think you will be able to see, quite clearly, the parts I have mentioned.

These then, are the appearances seen in a radiogram of the normal labyrinth. Before showing you radiograms of the abnormal labyrinth, I may state that I have only, so far, radiographed about 300 abnormal labyrinths, and what I am going to say about the variations from the normal, which I have seen in them, represents only my impressions of what I have seen in this number of cases. I cannot yet prove that these impressions are correct, and shall not be able to do so until sections of a pathological labyrinth which has been radiographed have been cut and examined microscopically. I can say, however, that at present one can distinguish the definitely normal and the definitely abnormal, and so far as I have gone, the great majority of cases with clinical signs show well marked changes in the bony capsule. But between these two extremes there is a group of cases in which one is suspicious that the labyrinth is abnormal; yet owing to the wide variations in the structure of the normal petrous bone, I cannot, as yet, be very definite in these cases. I hope, however, that in the course of time, and with added experience, and with the co-operation of other workers, that it will be possible to fill up this gap, and to show pathological changes in the internal ear with the same facility that one can show pathological changes in other bony structures of the body.

Radiogram 4, with 4a, is from a case showing clinical signs of early otosclerosis. If you compare this radiogram with a radiogram of a normal labyrinth, I think you will be able to

see a wide rim of porous, hazy bone extending from the neighbourhood of the oval window, round the basal and terminal one-and-a-half turns of the cochlea. The whole cochlea is hazy and ill-defined, and over the modiolus there is a rounded area of hazy, porous bone. In the diagram I have tried to show, as far as I can, these appearances, by shading. In connexion with otosclerosis, I may say that in the 300 cases of otosclerosis which I have radiographed, I have never seen the slightest sign of sclerosis in the capsular bone. The only pathological change which I have seen is one which would best be described as porosis, the capsular bone having a hazy and amorphous appearance. The best description I can think of to illustrate the changes which appear to me to take place in the capsular bone, is that they resemble those changes which are seen in other bones in the body in osteitis deformans. The capsular bone has that mortar-like, hazy appearance which is seen in osteitis deformans, and although, of course, I do not mean to imply that there is any connexion between the two conditions the appearances in the bone, as seen in a radiogram, are somewhat similar. In radiograms of otosclerosis I have noticed that these changes invariably affect the lower part of the vestibule, and basal turn of the cochlea, which is, I believe, in accordance with the changes seen microscopically. But I have also noticed that in advanced cases the capsular bone of the canals is similarly affected, and occasionally the bone surrounding the internal auditory meatus, especially along its lower border. I am aware that many otologists hold that otosclerosis does not affect the canals, but I can only say that in several cases of advanced otosclerosis I have radiographed, I have seen such changes, and I have never seen these appearances in a normal labyrinth. The radiogram just exhibited shows no such changes in the canals. The capsular bone is clear cut and well defined, and there is a definite abrupt line of definition between the capsular bone and the surrounding petrous bone.

Radiogram 5, with 5a, is one from an advanced case of otosclerosis; in it the hazy ill-defined cochlea is visible, but, in addition, I think it can be seen that the disease is also affecting the capsule of the superior canal. The bone is porous and hazy. It is densest near the membranous canal, and gradually shades off in density towards the periphery, there being no clear-cut line of definition between the capsular bone and the surrounding petrous bone. This is in marked contrast to the normal capsular bone, which is homogeneous in density throughout.

Radiogram 6, with 6a, shows Paget's disease affecting the labyrinth. You will see that in this condition the capsular bone is so completely atrophied that the capsule cannot be distinguished as such, and the only remaining normal part of the capsular bone is situated over the apex of the superior canal. Diagram 6a shows you the changes occurring in the labyrinth.

Radiogram 7, with 7a, is from a case of a tumour of the eighth nerve, which has destroyed the internal auditory meatus, and you can see the tumour projecting upwards above the superior border of the petrous bone.

Radiogram 8 is of another tumour of the eighth nerve which has entirely eroded the internal auditory meatus, and the bone in its neighbourhood.

Radiogram 9 is from a case in which a radical mastoid operation has been performed, and here you can see a large funnel-shaped opening into the apex of the external canal, which is due to a fistula. In the diagram 9a I have marked the fistula with the letter G.

I think these few cases are sufficient to illustrate the points which I have made, namely, that a radiogram can be taken to show the labyrinth, and that by radiography certain pathological changes can be shown occurring in the labyrinth. As I have already pointed out to you, these radiograms which are taken of a very small area situated deeply in the skull, necessarily lose a great deal of their definition and detail in reproduction; but I should be very pleased to show to any of you who are interested, the originals of these radiograms on a viewing screen, which will demonstrate the points I have mentioned, much more clearly than you can see them in a reproduced lantern slide.

II.—Mr. F. W. Watkyn-Thomas

said: I have two remarks to make before I say anything else. First, I must pay a tribute, not to Dr. Graham Hodgson's technical skill—the work he has shown us speaks for that—but to his untiring energy and enthusiasm; secondly, neither he, nor Mr. Jenkins, who introduces his paper with me, are in any way responsible for or perhaps I should say, guilty of complicity in, my conclusions.

As the majority of the cases on which I have been working with Dr. Graham Hodgson are cases of clinical otosclerosis I shall confine my remarks to this condition.

By "clinical otosclerosis" I mean cases in which there is an intact membrane, a patent Eustachian tube, a Rinne negative at 512 D.V. or higher, paracusis of Willis, and a negative Gelle. In all my cases except one there was a lowering of the high tone limit by air, as tested by monochord and Galton-Edelman whistle.

In all these cases there is a curious uniformity in radiological findings, a uniformity strangely at variance with the protean clinical manifestations. If these photographs are viewed from a distance of six feet or so it will be seen that in the otosclerotics the labyrinth stands out as a compact lump—the changes are not confined to one part of the labyrinth but affect the labyrinth capsule as a whole. I would emphasize "labyrinth capsule" because, however much I have suspected it, in no case have I proved, to my own satisfaction, any encroachment on the labyrinth lumen. The change, which can best be described as a "fuzziness" or swelling of the labyrinth wall, is specially marked in the region of the superior vertical canal and about the first turn of the cochlea; such changes in the region of the first turn—again without any narrowing of the lumen—are shown in one of the plates in Wittmaack's "Histological Study of Otosclerosis." Changes in the region of the internal auditory meatus are frequent, and here it does appear to me that, in some cases, there is a narrowing of the lumen.

An interesting point is the disparity between the clinical and radiological findings; for instance, in one case of mine the bone changes are much more advanced in the right ear than the left, but clinical examination shows that the left ear is deaf to the conversational voice at 8 in., while with the right ear the patient can hear at 18 ft. In another case, clinically unilateral, on the affected side there is nearly complete deafness to the conversational voice, but the high-tone limit on that side is normal for air and bone, although there is considerable change in the region of the first turn. This case is of particular interest when we recall the fact that, on the resonance hypothesis, the first turn is the part of the cochlea responsible for the acceptance of the high notes.

Another point of interest is that the bony change is confined to the labyrinth and follows the line of the capsule, there is no diffusion into the surrounding petrous, except at one spot, and that is the angle between the superior canal and the free border of the petrous; in many of these skiagrams an extension of the bone change can be seen in this region.

A third point is that, in none of these skiagrams of otosclerotic patients is there any evidence of true sclerosis. The labyrinth capsule is thickened and expanded, but, if new bone has been laid down, it would appear to be spongy bone, not compact.

At this stage it would be premature to attempt any deductions; much more work must be done, and radiological and histological findings must be correlated in a series of cases. It may, however, be justifiable to hazard two suggestions; neither is original. Manassez regards the nerve-deafness of otosclerosis as the primary lesion; the bone changes as secondary. If, as these skiagrams suggest, otosclerosis is a disease of the whole of the labyrinth capsule, it is easy to see how, in the irregular spread of the disease, any nerve fibre group might be picked out.

Again, it must be admitted that there is one hypothesis as to the pathogenesis of otosclerosis which certainly does fit in with the radiological findings. The labyrinth is in the petrous, but not of it. The labyrinth develops independently, and its capsule is formed in two layers, a layer from the mesoderm around the primitive auditory capsule, and an outer layer from the muco-periosteum of the tympanic cavity and from the dura. Between these two layers there is, in the fœtus, a lymphatic space, and the suggestion has been made that otosclerosis is due to an infection of this space, which has not been perfectly obliterated.

Mr. ALEX. R. TWEEDIE said that Mr. Watkyn-Thomas had referred to the high tone limit for bone conduction on one side, and he (the speaker) would point out that as we had no noise apparatus for obliterating this function on one ear, it was impossible to decide accurately by which ear bone-conduction of the high tone limit was really heard. He himself was endeavouring to have a noise apparatus made which would exclude the appreciation for the high tones on one side.

The Saccus Endolymphaticus and an Operation for Draining for the Relief of Vertigo.

By Professor GEORGES PORTMANN (Bordeaux).

THE contribution to the therapeutic of vertigo which forms the subject of this work establishes the practical result of a series of researches of compared anatomy, human anatomy, and physiology on the saccus endolymphaticus, that I have pursued incessantly for the last eight years.

The study of anatomy had shown me the constancy and the considerable dimensions of the saccus endolymphaticus in the vertebrates and, specially, in man.

Actually an intracranial extension of the labyrinth, lying on the postero-superior surface of the petrous portion of the temporal bone, in a depression which I have called the "fossa endolymphatica," the sac is hollowed in the thickness of the dura mater, the division of which into two forms an antero-inferior wall, juxtapetrous, placed against the bone, and a posterior superior wall, cerebellar, in relation with the arachnoid and cerebellum.

The enormous vessel, namely, the lateral sinus, skirts half its outer circumference and is only at this level separated from it by some connective layers.

Lastly, the saccus, in direct relation through the ductus endolymphaticus with the membranous cavities, utricle, and saccule, contains a clear fluid which is simply endolymphatic fluid.

In October, 1922, I presented before the International Congress of Otology some of my experiments on fish, in which I endeavoured to prove the importance of the endolymphatic organ in auricular physiology. Since then I have continued fresh researches in order to confirm those former results and I believe that at the present moment the functional importance of the saccus endolymphaticus is no longer contested by anyone.

In man, in whom anatomical conditions are widely different, and in whose case only physiology and pathological anatomy can enlighten us, we are right in thinking that this saccus owing to its place in the thickness of the dura mater is in continual pressure against the subarachnoid spaces.

The modification of the tension of the spinal fluid is thus liable to disturb the normal function of the labyrinth. This is a question that is beginning to occupy the minds of some of the researchers, and in January, 1925, Georgio Ferreri published in the *Revista oto-neuro-oftalmologica* the result of his work on the action of the experimental hyperpressure of the spinal fluid on the posterior labyrinth. Quite recently (in June, 1926), and after the results I had obtained with regard to fish, W. J. M'Nally has, in the Pharmaceutical Laboratory of the University Rijki at Utrecht,[1] undertaken a series of experiments on the rabbit, in which he provoked this increase, and the lessening of pressure on the level of the saccus endolymphaticus.

If he has not noticed any labyrinthine disturbances in producing a hyperpressure on the saccus (but on this subject further experiments seem necessary, as a control), the opening of the saccus, through its causing the issue of the endolymphatic fluid, has invariably produced a lessening of the *tonus* of the limbs on the same side.

A phenomenon, superposed on the lessening of tonus of the limbs, which, as Magnus and de Kleyn have proved, is produced after an opening of the membranous labyrinth, is produced after section of the eighth cranial nerve.

These facts having been admitted allow of our comprehension of the importance of the modifications of pressure either of intracranial, that is to say extralabyrinthine, or endolabyrinthine origin, that may support the saccus endolymphaticus and the consequences that may result.

In this first case, the saccus may be compressed by the pathological modifications affecting an adjacent organ, the lateral sinus, or rather the meninges and cerebellum. The

1 W. J. McNally, "Experiments on the Saccus Endolymphaticus in the Rabbit," *Journal of Laryngology and Otology*, June, 1926.

increase of pressure produced by means of the saccus endolymphaticus on the intrapetrous labyrinth may give rise to Ménière's syndrome: vertigo, deafness, buzzing. It therefore appears to be logical to incriminate the saccus endolymphaticus as the vestibular nucleus when these arise during the course of an intracranial hyperpressure, whatever may be the cause of such symptoms.

In the second case the increase of endolymphatic pressure may proceed from an endolabyrinthic cause, and a long while ago attention was drawn by otologists to "serous labyrinthitis"; the increase of the endolymphatic fluid sets up in the cochlear, otolithic and vestibular nerves, functional trouble which evokes the appearance of the triad of Ménière—thus realizing a true "auricular glaucoma."

There exists elsewhere more than one analogy between intra-ocular hypertension and endolabyrinthine hypertension of serous origin. In one case, as well as the other, the question is most often one of crises of localized hyperpressure occurring in subjects of high tensions or subjects with a hyperexcitable sympathetic system.

The recent and very interesting researches performed by Guild, in the Department of Anatomy, University of Michigan, and presented in May, 1927, before the American Otological Society in New York, show us the importance of the saccus endolymphaticus as an organ of filtration of the endolymphatic fluid. It serves as the principal place of outflow of the endolymph; not, as some have thought, as the point of origin of endolymph. The escape of fluid is not through openings in the wall, but by direct passage through the layers of the wall, being thus comparable to the flow of cerebro-spinal fluid through the arachnoid villi. Then in some cases the possible interpretation of abnormal distention of the membranous labyrinth (auricular glaucoma) as an impairment of the normal pathway of the endolymph can be discussed.

Even, to continue our comparison, as in glaucoma the ophthalmologists puncture the cornea in order to suppress the intra-ocular hypertension, capable of destroying for ever the value of the eye, so it seems logical in some cases of serous labyrinthitis, when medical treatment has failed, to make a decompression of the internal ear by the removal of the excess of the endolymphatic fluid.

Then the membranous labyrinth protected in the heart of this thick, strong box, that is the bony capsula of the labyrinth—separated even from this bony capsule by the perilymphatic spaces, is practically inaccessible and safe from destruction. At one point only it is vulnerable, namely, at the level of the endolymphatic sac, placed in the fossa which bears its name, on the posterior surface of the petrous bone. It is there that it is well to carry out a decompression operation on the labyrinth.

In this way the otologist obtains a new perspective of surgical therapeutics in those cases in which one might suspect the existence of a serous labyrinthitis, or in which a cranial hyper-pressure, whatever the cause, will produce a violent reaction of the labyrinth.

My operation consists in reaching the fossa endolymphatica and in discovering the saccus in order to open its petrous wall. An exact knowledge of the relation of the fossa endolymphatica is indispensable. Let us recollect that, situated on the level of the posterior surface of the petrous bone, this triangular fossa, at the supero-internal angle of which opens the aqueductus vestibuli, is in external relationship with the lateral sinus which convolutes its outside and lower part before projecting itself into the jugular bulb at the lever of the outside portion of the posterior lacerate foramen.

It is therefore the external third portion that would be the easiest route of access to the fossa endolymphatica, the lateral sinus being in fact the most invariable landmark. On the other side, the fossa extends to 5 mm. anterior to the vertical portion of the aqueductus Fallopii. By starting from the internal border of the lateral sinus, but remaining at the level of the intersinufacial zone, one reaches the fossa.

In a word it will be necessary to search for the saccus through the mastoid in the area of a triangle formed, above, by a line corresponding to the lower surface of the antrum; in front, by the aqueductus Fallopii, behind, by the lateral sinus.

Operative Technique.

First step.—Incision to the bone at the level of the retro-auricular groove. Exposure of the surface of the mastoid; one then discovers the spine of Henlé and the external petrosquamous suture.

92 Portmann : *Saccus Endolymphaticus*

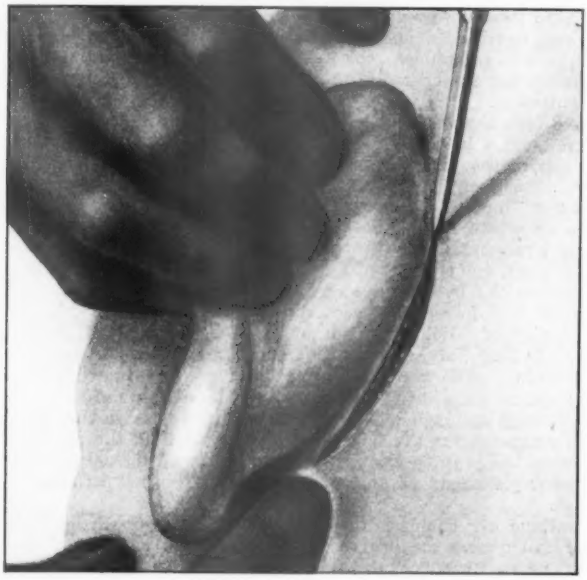

FIG. 1.

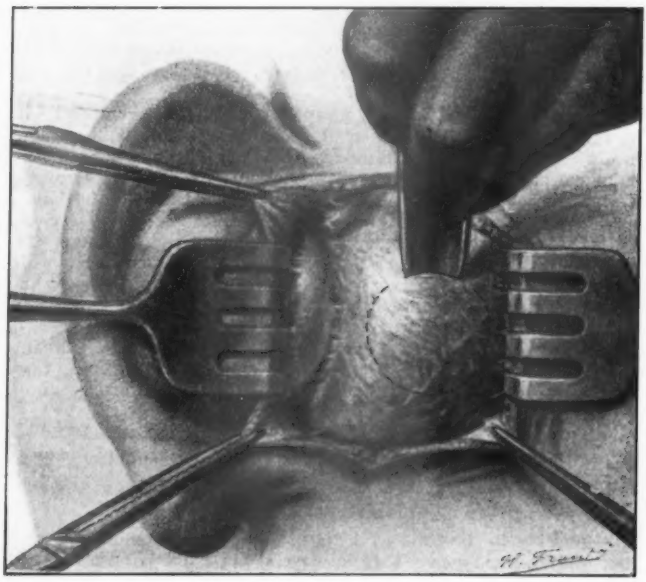

FIG. 2.

Second step.—Trepanning of the mastoid at the level of a square of attack situated lower down than that of the classic trepanning area for mastoiditis. The first stroke of the gouge is made on a horizontal line passing under the spine of Henlé. The second in front, on a vertical line situated 3 mm. behind the posterior edge of the external auditory meatus. The third is made horizontally at 1 cm. below the first. These three first strokes of the gouge are made perpendicularly on the surface of the bone; for the fourth, on the contrary, the instrument is held obliquely on the bony surface, 1 cm. behind the second. In gouging out this deeply, one finds the lateral sinus, the true landmark of the operation.

This "square of attack" has as its aim to approach the lateral sinus without opening the antrum, thus keeping the operation as aseptic as possible, without extensive communication with the middle ear.

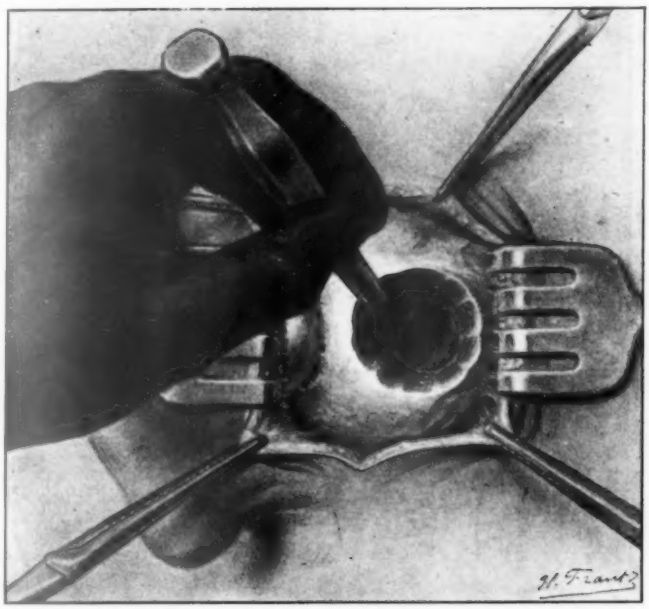

FIG. 3.

Third step.—The bony wall of the sinus having been scraped and its internal part removed, one detaches with a blunt elevator, as delicately as possible, the dura mater which covers the posterior surface of the petrous bone, inclining inside and slightly above, at a length of about 3 to 4 mm. A dura mater protector is then put in place, for it is now indispensable to divide with a fine graving tool the bony region which represents the most external part of the fossa endolymphatica. Recollect at this moment that the aqueductus Fallopii is near, and that the facial nerve could be injured by an unlucky stroke of the operator.

This bony region having been ablated, one again unfolds the dura mater, inclining always inside and slightly above until one comes to a zone of closer adhesion of the dura mater at the bone and a slight bony dip. We have now arrived at the level of the supero-internal part of the fossa, at the exact point where the aqueductus vestibuli begins.

Fourth step.—We have freed the petrous wall of the saccus endolymphaticus by removing the greater part of the fossa endolymphatica. By the help of a very fine needle, mounted on

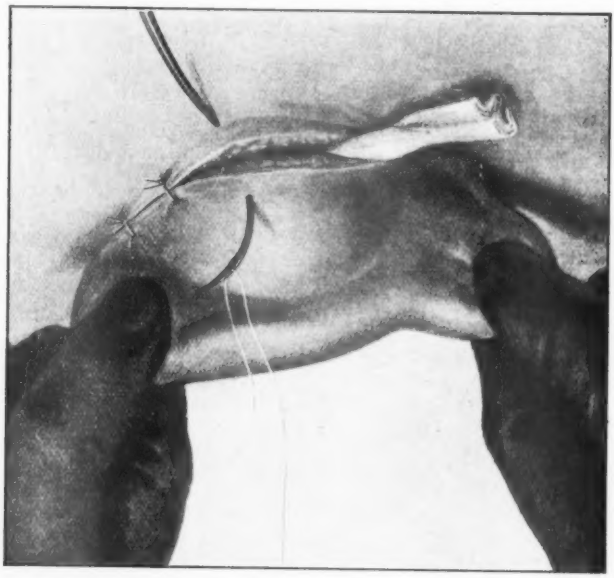

FIG. 5.

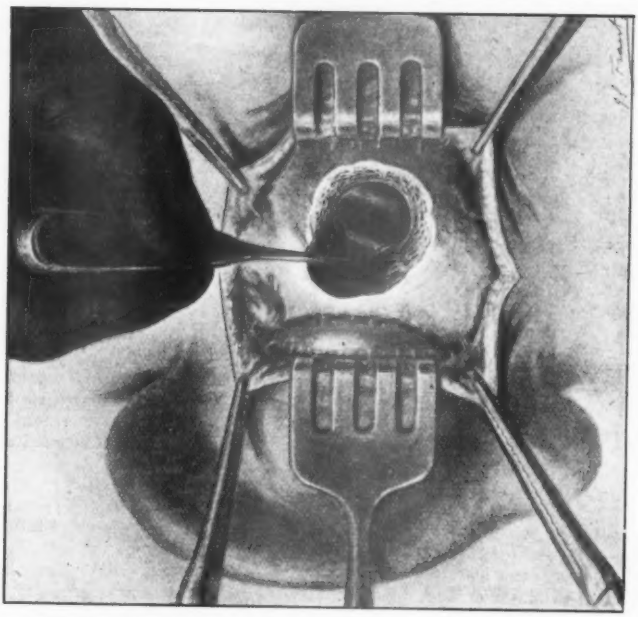

FIG. 4.

a Luer syringe, one makes an exploratory puncture of the saccus, *in contact with the zone of adherence*. The operator, who feels perfectly the sensation of penetration in the little cavity of the saccus, then makes, with a paracentesis knife, an incision of from 2 to 3 mm. at the most in the saccus. One or two drops of fluid, like spring water, flow from the opening of the wound.

The operation now being over, the retro-auricular wound is sutured. Slight drainage is rendered possible by the insertion of a piece of gauze, in the event of any considerable bleeding in the course of the bony trepanning.

It is a particular point of this detail in technique that the puncture, just as much as the incision of the saccus, ought to be made, as far inside as possible, near the zone of the adhesion.

It should be recollected, in fact, that the saccus which is occasionally atrophied in old age, would be reduced only at its juxta-canicular part so that, in making an incision far from the zone of adhesion, one would risk reaching only the meninges and missing the saccus. By operating at the contact of the bony wall which constitutes the supero-internal part of the fossa, one is sure to reach it even if it were much atrophied.

This operation, though a delicate one, is generally comparatively simple, as I have shown in my Clinic at the University of Bordeaux before the Members of the Sixth Meeting of the Visiting Association of Ear, Nose and Throat Surgeons of Great Britain in April, 1927. To me it seems to be the method of choice for reaching the saccus endolymphaticus and appears to be full of promise for the surgical therapeutics of a great number of labyrinthine diseases, one symptom at least of which, namely, the vertigo, is particularly painful to the patient.

I have on several occasions performed this operation most successfully, and quite recently (October 10, 1926) I reported (1) at Gröningen, before the International Oto-Rhino-Laryngological College, and (2) at Rome (October 23, 1926) before the Italian Congress of Oto-Neuro-Oculists—two typical cases of vertigo, cured by opening of the saccus endolymphaticus. The severity of the vertigo had prevented the unhappy patients from doing any kind of work.

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The Formation of a Circumscribed Interdural Abscess at the Site of the Saccus Endolymphaticus.

By Jobson Horne, M.D.

It is nearly thirty years since I recorded a case of pus from the middle ear finding its way into the cranial cavity along the route of the ductus and saccus endolymphaticus. Since then I have had only one other opportunity of verifying the occurrence post mortem.

The post-mortem researches represent the practical side of the question, therefore this occasion seems appropriate for a demonstration of specimens illustrating the morbid process. Owing to the rarity of the condition there must be many present who have not seen similar specimens.

Pus, when pent up within a labyrinth, the outer walls of which are intact, presumably follows the line of least resistance in endeavouring to escape into the cranial cavity. This is not necessarily by way of the internal auditory meatus. Another route, which at times apparently offers less resistance, passes along the aqueductus vestibuli to a fissure on the posterior aspect of the petrous bone. Through this fissure the prolongation of the membranous labyrinth, known as the saccus endolymphaticus, originally passed, and in adult life it *may* remain sufficiently patent to permit the passage of pus. A shallow depression on the posterior surface of the bone below the orifice of the aqueduct and immediately above the lateral sinus marks this natural exit.

The development of a circumscribed abscess at this spot is well demonstrated by the two specimens which form the basis of this communication.

The first specimen was exhibited in the museum at the Sixth International Otological Congress held in London, August, 1899. The clinical notes of the case will be found in the *Transactions*.[1] At the time of compiling the descriptive catalogue of the museum of the Congress[2] no similar specimen had been found in the museums of morbid anatomy attached to the hospitals in the metropolis or provinces. It follows, bearing in mind the relative frequency of intracranial complications of suppurative disease of the middle ear, that this particular morbid process must be of rare occurrence. Curiously, within twelve months the second specimen was met with.

Photographs of the two specimens are reproduced. In the first the dura mater is left *in situ*, and shows the appearance of the abscess sac from the cranial aspect. In the second, the dura mater is reflected, and with it the abscess sac, to show the line of potential communication between the internal ear and the posterior fossa.

The *first* specimen is the right temporal bone of a lad aged 19, who died from lateral sinus thrombosis and meningitis. Post mortem, a slight amount of meningitis was found over the pons and medulla, more marked over the right than the left side, and the serous fluid beneath the tentorium was increased. In the neighbourhood of the mastoid cells on the right side, beneath the dura mater, and almost surrounding the lateral sinus, was a collection of pus—about three-quarters of a drachm. No pus was found beneath the dura mater over the middle ear. In the lungs there was some old tubercle at both apices; at the right apex there was one small cavity containing pus. Bacterioscopic examination of the pus from the middle and internal ear yielded a diplococcus as well as staphylococci and streptococci.

Dissection of the temporal bone revealed the following: Upon removing the anterior wall the posterior wall was found to be eroded, Shrapnell's membrane perforated, and the

1 *Transactions of the Sixth International Otological Congress*, London, August, 1899, p. 392, *et seq.*

2 Descriptive Catalogue of the Museum of the Sixth International Otological Congress, p. 100, No. 695.

incus lost. There was considerable destruction of the outer attic wall up to the roof of the middle ear.

In the middle fossa could be seen some localized pachymeningitis over the roof of the middle ear, which was an irregular carious area and perforated.

In the posterior fossa, immediately beneath the superior petrosal sinus in the neighbourhood of the site of the saccus endolymphaticus, and stretching across the lateral sinus (as seen in fig. 1), was an abscess sac. The incision through the abscess shows that its walls are *between* the layers of the dura mater. The abscess has perforated in two directions; there is a perforation (through which a bristle passes) into the posterior fossa, which caused posterior leptomeningitis, and on opening the lateral sinus it was found that the abscess had perforated the wall, causing septic thrombosis of the sinus from that point downwards.

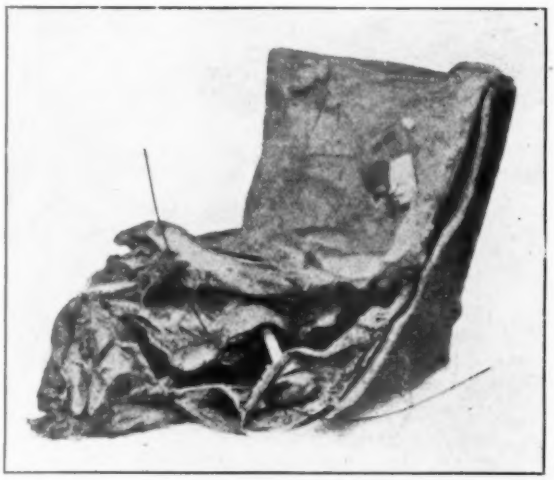

FIG. 1.—POSTERIOR PART OF RIGHT TEMPORAL BONE.

The lateral sinus is laid open in its entire extent. A white rod stretches across the sinus and lies within the abscess sac. A bristle passes through the perforation in the posterior wall of the sac, through which pus escaped into the posterior fossa. Another white rod is placed in the internal auditory meatus.

On reflecting the dura mater from the internal auditory meatus, pus was met with surrounding the seventh and eighth nerves. The labyrinth presented through the bone a yellow appearance. The roof of the labyrinth was dissected out, and the internal ear was found to be occupied throughout its entire length with dark-brown pus.

Infection had spread from the middle ear to the labyrinth, via the oval window; and by stripping the dura mater from above downwards to the aqueductus vestibuli, it could be seen that pus had emerged along the aqueduct to the site of the saccus endolymphaticus.

The *second* specimen is the right temporal bone of a man aged 37, who also died from lateral sinus thrombosis and meningitis. Post-mortem, the basal aspect of the pons, medulla, cerebellum and region of the optic tracts, and the surface of the spinal cord, were covered with a layer of greenish fibrinous lymph mixed with a quantity of purulent matter. The amount of fluid about the base of the skull was much increased.

In the posterior fossa of the skull on the right side, and between the posterior aspect of the petrous portion of the temporal bone and the superjacent dura mater, there was a

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flattened abscess sac which held about a drachm and a half of pus. The sac communicated by a small gap in the dura mater with the cranial cavity. The lateral sinus contained an ante-mortem clot.

The temporal bone was removed for further examination. It was found that the antrum had been opened, and that the membrana tympani and ossicles were absent. On reflecting the dura mater downwards and inwards from the middle fossa, no localized pachymeningitis was present over the roof of the middle ear, which was intact; and no pus was present in the internal auditory meatus. The labyrinth as seen through the bone looked yellow. The internal ear contained traces of dark-brown pus, and was carious.

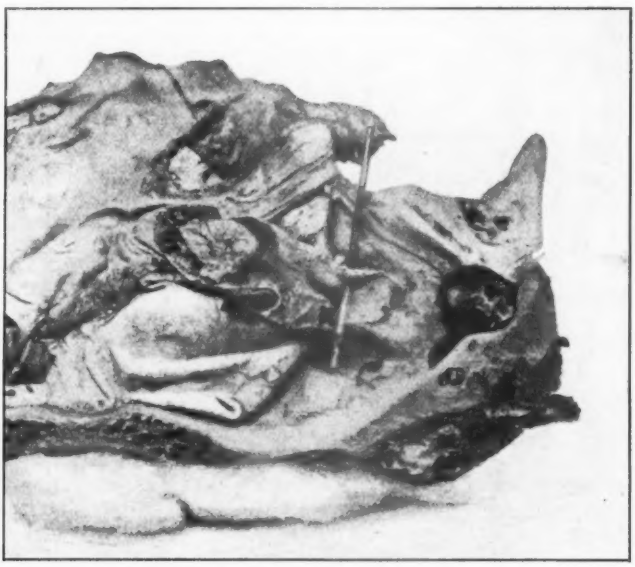

FIG. 2.—POSTERIOR PART OF RIGHT TEMPORAL BONE.

The dura mater has been reflected inwards; the petrous portion of the bone and the groove of the lateral sinus laid bare. A glass rod is placed between the position of the aqueductus vestibuli and the internal auditory meatus, and across the rod stretches that part of the dura mater which is more intimately adherent to the bone at the site of the saccus endolymphaticus.

Upon stripping the dura mater farther downwards to the level of the exit of the aqueductus vestibuli, it became evident—as can be seen in fig. 2—that pus had tracked within a cord-like prolongation of dura mater, which enters a fissure in the bone at the site of the orifice of the aqueduct and the saccus endolymphaticus, and, moreover, that the abscess sac above referred to was formed *between the layers of the dura mater, and not between the dura mater and the bone.* The abscess sac is shown reflected, together with the dura mater, inwards and immediately to the inner side of the glass rod.

Both patients had been subject to suppuration in the middle ear since early childhood. They had both been in good health, and followed their occupations up to the day of the fatal extension, which was marked by giddiness; in the first case thirteen days, and in the second twenty-four days, before death.

A knowledge of the lesion that has been described is, I think, of utility to the aurist and of interest to the morphologist. Such an abscess is within reach of surgery, and this gives a practical bearing to the recognition of its possible occurrence in cases with symptoms pointing to intralabyrinthine suppuration.

A question of morphological interest is, what part, if any, the saccus endolymphaticus can take in developing the walls of the abscess. Does the pus accumulate within the saccus itself, which by distension becomes converted into an abscess sac, or does it accumulate between the two layers of dura mater which originally received the saccus endolymphaticus?

The saccus endolymphaticus in the human fœtus is so minute that even in the earlier months it is difficult to demonstrate it by dissection; its whereabouts are indicated by a varying degree of looseness of the folds of dura mater covering the posterior surface of the petrous bone at its site. I am therefore inclined to the view that an abscess is not contained within the saccus itself, but between the two layers of dura mater which originally contained the saccus. After fœtal life, and the disappearance of the saccus, these two layers continue to form a potential cavity. A cord-like prolongation from these layers is carried into the fissure immediately over the orifice of the aqueductus vestibuli, along which communication may be opened up from the internal ear. Complete closing of the fissure would cut off this communication, but it does not appear that the closing necessarily occurs with growth. The saccus endolymphaticus may, however, be regarded as another instance of how the vestigium of an embryonal structure may become a source of danger to the organism.

Discussion.—Dr. DAN MCKENZIE (President), in thanking Professor Portmann on behalf of the Section for his beautiful and graphic demonstration, said that Members would look back on that evening as marking a distinct advance in the possibilities of aural surgery. He wondered whether the method might not be adopted experimentally, not only in the treatment of vertigo, but also perhaps in some cases of nerve deafness.

Sir CHARLES BALLANCE said that Professor Portmann's demonstration was based on just that kind of investigation to which great scientific problems lent themselves, and which, when carried out in this way, generally led to some practical result.

Without doubt the anatomical and physiological investigations of Professor Portmann had borne fruit in the discovery and description of an operation which might prove of the greatest service of mankind.

Fortunately the region in which this remedial operation had to be performed was well known to surgeons and otologists. The region, the outer part of the posterior surface of the petrous bone had been often exposed by many; he had often exposed it for the purpose of draining an abscess in that situation. Only a few months previously he (Sir Charles) had watched with great interest a splendid operation performed by the President in a condition in which the saccus endolymphaticus was involved. Dr. McKenzie had followed a tract of disease through very hard bone, leading on to the posterior surface of the petrous bone in the region of the saccus.

The whole address had been of extreme interest, as it opened with researches upon the lower animals, presenting a continuous picture from the selachiæ right up to man. That was the foundation of what could be looked forward to in the treatment of tinnitus and vertigo, and—he hoped with the President—of deafness as well.

In this operation Professor Portmann was probably on the verge of further discoveries, and he (Sir Charles) hoped that at some future date the Professor would be in a position to indicate exactly the cases in which the operation could be performed.

Mr. G. J. JENKINS said that the term "glaucoma" had been used by him in this connexion in 1913. He had opened the internal semicircular canals to relieve vertigo, but had not felt sure of his ground. Mr. Mollison also had published three or four cases in which he had opened the external semicircular canal for the relief of vertigo. He found accumulations of

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cerebro-spinal fluid in front of the cerebellum in some cases of vertigo, and it had been suggested that these might be cases of dilated saccus endolymphaticus. The difficulty would be in deciding in which kind of case of vertigo the operation suggested should be done.

Mr. SYDNEY SCOTT said that Professor Portmann's demonstration would encourage otologists to pay more attention to the saccus endolymphaticus.

As a practical outcome of Dr. Jobson Horne's demonstration in cases of labyrinthitis should we be content with opening and draining the labyrinth or should we also expose the dura mater, in the way Professor Portmann had described, and open up the saccus endolymphaticus?

In the great majority of cases of vertigo it was quite unnecessary to open the labyrinth or the saccus. In the operations designed to give relief in specially selected cases the middle ear was opened to obtain access to the labyrinth.

When Professor Portmann performed his operation on the saccus he (Mr. Scott) suggested he should try to do so without opening mastoid cells and so avoid possibly fallacious conclusions that it was only drainage of the saccus which caused the beneficial results.

Dr. J. S. FRASER said that he regretted that Dr. Albert Gray was not present, as he had worked along much the same lines as had Professor Portmann, e.g., post-mortem injection of indian ink into the saccus endolymphaticus, causing the ink to circulate throughout the entire membranous labyrinth.

How did Professor Portmann explain the good hearing of patients suffering from meningitis? He had shown the relation of the saccus endolymphaticus to the posterior fossa of the skull and had explained that changes of pressure in the posterior fossa affected the labyrinthine pressure. The pressure was, of course, greatly raised in meningitis and yet patients suffering from meningitis heard remarkably well.

With regard to "glaucoma of the labyrinth," he (the speaker) thought there must be some disease in the inner ear corresponding to glaucoma in the eye. Here we had to do with the secretion and circulation of the endolymph. The endolymph was probably secreted by the cells of the stria vascularis. Any blockage of the endolymphatic duct or sac would be likely to cause "glaucoma of the labyrinth." In his (Dr. Fraser's) own case of congenital syphilis affecting the middle and internal ear, it was true that the ductus endolymphaticus was blocked up, and that the utricle, saccule and cochlear canal were dilated, but it must be remembered that one met with dilatation of the membranous labyrinth not only in cases of blocking of the ductus endolymphaticus but in many cases of spontaneous cure of labyrinthitis, in which no blockage of the endolymphatic drainage could be found. There must be some other causes of dilatation of the membranous labyrinth than mere blockage of the ductus and saccus.

With regard to empyema of the saccus endolymphaticus, he had microscopically examined over 100 cases in which the patient died as a result of the intracranial complications of middle-ear suppuration. In thirty-five of these the labyrinth was involved, but in only one case was there empyema of the saccus endolymphaticus. That was in opposition to what Mr. Sydney Scott had said. Inflammation in the perilymph spaces was common, and in a smaller number of cases there was inflammation of both endo- and perilymph spaces, but affection of the endolymph spaces alone was very rare if not unknown.

Concerning experimental work on the labyrinth, he was sure Members would remember Professor Biehl's work which was published in the *Journal of Laryngology* a short time ago. Biehl made an incision in the tympanic membrane in the lower posterior part, and then inserted an angled needle round the niche of the round window, and tapped the peri-lymph space at the beginning of the cochlear canal. Biehl was doing this operation for the same class of case as that described by Dr. Portmann. The great difficulty was to know how one could be sure that the vertigo was due to hypertension in the inner ear.

The operation proposed by Dr. Portmann was not unlike that which was described for exposure of the jugular bulb from the mastoid wound. He (the speaker) admitted that in this operation there should be no injury to the posterior semicircular canal, but it might be a delicate matter to avoid such injury.

Mr. H. J. BANKS-DAVIS said he presumed that Professor Portmann closed up the wound completely. He (the speaker) did not understand what was the purpose of the operation if

its result was to be only temporary. Did a fistula remain in the saccule, or was it merely opened and allowed to close up? Also, was drainage subsequently carried out?

Mr. A. R. TWEEDIE said that when he first heard Professor Portmann's demonstration last October, he then expressed the opinion that it would provide a true explanation of the cases, to which Bárány drew attention, of suspected cerebellar lesions, in which, however, when the posterior cranial fossa was opened nothing abnormal was found, whilst the patients subsequently recovered.

Bárány did not agree with this view, but he (Mr. Tweedie) maintained that this operation did re-establish the secretory connexion between the saccus endolymphaticus and the dural spaces, which he (the speaker) submitted was the essential objective of the operation.

He was pleased to hear stress laid upon the need for a careful selection of cases for the operation in question. It was, of course, important to try other methods first, reserving operation as a last resort. A possible indication, he thought, for this operation, might be a hyposensitive response to the galvanic and caloric tests, which might be regarded as the result, possibly of hypertension in the labyrinth.

He was very glad that Dr. Jobson Horne had brought his specimen forward, as one of his (the speaker's) earliest otological recollections was of a demonstration at St. Bartholomew's Hospital, by Sir Frederick Andrewes, who drew attention to the saccus lymphaticus, and the part it might play in the production of meningitis.

Professor PORTMANN (in reply) said he was interested in hearing that the term "glaucoma" had already been used in this connexion; it was often the case in medicine and surgery to find that one's supposed originality had been anticipated. Bárány had said it was not necessary to open the saccus endolymphaticus, and that he had had good results from exposure of the dura. But, even so, Bárány performed the same operation as himself (Professor Portmann), because he detached the dura from the petrous bone and thus ruptured the ductus endolymphaticus when this organ penetrated into the aqueductus vestibuli. He agreed as to the importance of what had been said relating to the posterior semicircular canals, but with care in the operation they could be avoided. He much preferred the route of entry which he had described.

When a new surgical procedure was initiated it could not be expected to be perfect at once. It was temporary, but it might be the means of making a way for a new communication. If closure occurred and a year or two afterwards there was a return of the symptoms, the operation could be repeated. It was not yet possible to know whether troubles were set up by pressure irregularities, but when a case appeared to resist all medical treatment, this operation might well be tried.

Dr. JOBSON HORNE (in reply) emphasized the importance of bearing in mind that the saccus endolymphaticus in the human subject very seldom persisted into adult life, and therefore the results of an operation in search of the saccus endolymphaticus for the relief of vertigo must be highly speculative.

(1) Deafness due to Occlusion of the External Auditory Meatuses Caused by External Otitis. Operation to Restore Patency of Left Meatus Resulting in Greatly Improved Hearing.

By T. B. JOBSON, M.D.

C. B., MALE, aged 31, a railway fireman. First seen March 1, 1923.

External otitis, right and left, walls of meatuses uniformly thickened—some pasty debris scooped out—treated with various lotions and drops without benefit. Seen again 1926: local treatment and vaccines. No improvement.

January 18, 1927.—Operation: mastoid incision, pinna reflected forward, cartilage of meatus separated, strip of cartilage removed from posterior wall, bony meatus

enlarged by removal of some of posterior wall; rubber tube 1 cm. diameter left in for ten days.

(It is important to keep the rubber tube in for about ten to fourteen days, as there is a great tendency for the meatus to contract as it heals.)

Hearing for watch, which before operation was only "contact" right and left, is now 6 in. on left. His hearing for conversation is now good. Before operation it was only 2 ft.

Sir JAMES DUNDAS-GRANT said such cases of occlusion could sometimes be successfully treated by means of the laminaria tangle tent. In one case in which the removal of hyperostosis of the meatus by another surgeon was followed by complete occlusion he (Sir James) forced the point of a probe through the fibrous tissue and dilated the minute opening by means of a fine tangle tent whittled down to a point. He then introduced a minute rubber drainage tube, and eventually, after numerous dilatations and the introduction of rubber tubes of greater and greater size, the passage became so large that there was scarcely any narrowing. In contractions following the radical mastoid operation he had effected considerable widening in a similar way. In addition he had plugged the outer part of the cavity with cotton wool soaked in Burow's solution of acetate of lead and alumina acetate, and the result was excellent.

Mr. A. D. SHARP said he had obtained excellent results in such cases by a plug of 10 per cent. ichcthyol in glycerine. Even the very narrow type responded excellently.

Aural Electrodes for Use in the Treatment of Chronic Otorrhœa by Zinc Ionization and Zinc Electrolysis.

By A. R. FRIEL, M.D.

Dr. A. R. FRIEL exhibited some electrodes for the ear which he uses in the treatment of chronic otorrhœa.

(1) An electrode for the treatment of sepsis in an accessible position.

(2) An electrode consisting of two zinc wires fastened to a light wooden handle, for the destruction of polypi or large granulations; and also for the treatment of an annular stricture of the meatus.

(3) A monopolar zinc electrode for the incus area; and a bipolar electrode for the same area. Both of these are used to destroy tissue in order to make an inaccessible area more accessible.

(4) A bipolar electrode for the destruction of part of the outer wall of the attic.

Dr. NORRIE (of Calcutta), introduced by Dr. Friel, exhibited two portable rheostats and electrodes for the treatment of suppuration in the ethmoid cells: nine needles for the treatment of hypertrophy of the inferior turbinals: and the electrode which he uses in the treatment of chronic otorrhœa. He showed an ingenious arrangement for retaining an electrode in the position in which he had placed it.

Three Cases of Deafness.

By H. NORMAN BARNETT, F.R.C.S.

Case I.—MRS. J., aged 44, female.

History.—No deafness in family. One brother had tuberculosis. Patient had measles and scarlet fever as a child but no history of ear trouble. Has been deaf since 1914, getting steadily worse since then. Occasional tinnitus of a mild character.

Examination.—Septic tonsils, deflection of septum, retracted tympanic membranes.

Hearing: Right B. C. – 2; R. –; T. F. (C.256) – 30; Conversation 1 ft.; Whisper 6 in.; Watch 2/160. Left B. C., N.; R. – ; T. F. – 40; Conversation 2 in. Whisper 1 in.; Watch P./160.

Treatment.—Enucleation of tonsils, submucous resection of septum, submucous fracture of posterior ends of turbinate bones. Ionization over mastoid and vaporization through Eustachian catheter with warm iodine, camphor and carbolic vapours. Oto-massage.

This case is still under treatment and is improving, but improvement is much slower than in the other case reported as still under treatment. The patient can now hold conversation with one person at a 3 ft. radius. The case will require a considerable time to complete.

Case II.—W. G. J., aged 35, male.

History.—In 1916 was blown up by shell. Part of shell hit steel helmet on left side, indenting it badly. Patient was unconscious for twelve hours. When consciousness returned he noticed he was deaf on left side. This deafness partly passed off later but hearing was never normal. Two years ago deafness got worse and gradually increased. Two months ago had influenza and since then the hearing on the right has been affected and has become steadily worse.

Examination.—Showed septic tonsils; deflection of nasal septum. Tympanic membranes appeared normal.

Hearing.—Right: B. C. – 1; R. – ; T. F. – 30; C. 6 ft.; Wh. 2 ft.; W. 6/160. Left: B. C., N.; R. – ; T. F. – 40; C. 2 ft.; Wh. 1 ft.; W. 4/160.

Treatment.—Enucleation of tonsils, submucous resection of septum, submucous fracture of posterior ends of turbinate bones, ionization over mastoid and vaporization through Eustachian catheter with warm iodine, camphor and carbolic. Oto-massage.

This patient is now under treatment and steadily improving; he is able to perform his duties without difficulty. The case will probably require another fortnight or three weeks to complete the cure.

Case III.—Dr. T., aged 50, male.

History.—General health poor; general family health good; no other member of family deaf.

Query as to middle-ear disease following measles at four. At thirty, while starting motor-cycle, suddenly got a "stuffy" feeling in ears which lasted for about eight years. While serving in Aden the ears seemed to be better but not normal. Had malaria badly in 1915, and has had twelve relapses—blood normal for past few years and no quinine during that time. Also had dysentery in 1915. Was deafer two months after this.

History of Deafness.—Has never heard as well as other people, but noticed deafness particularly as a student when using stethoscope. Deafness became worse about eighteen years ago. Four years ago saw a specialist, who suggested operation on turbinates, which was carried out with improvement for a time. Two years ago began to get steadily worse and had great difficulty in using stethoscope, and in September, 1925, the left ear became completely deaf. At this time patient had some inflation treatment with little or no result. Was advised to give up practice as nothing could be done for his hearing.

Examination.—December 22, 1925. First seen by exhibitor. Marked deflection of the nasal septum with an adhesive remnant of the right middle turbinate. Had buried septic tonsils and retracted and inflamed tympanic membranes. Antra clear on transillumination.

Hearing.—Right: B.C., N.; R. –; T. F. – 20; H. N. – 10; C. 20 in.; Wh. 20 in.; W.P/60. Left: B.C., N.; R. –; T. F. – 15; H. N. – 15; C. 18 in.; Wh. 15 in.; W.P/60.

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Treatment.—Submucous resection of cartilage and bone of nasal septum which was found to be fractured; removal of diseased and adherent tonsils; administration of ultra-violet rays, general and localized for throat. Subsequently oto-massage with ionization over mastoid and vaporization through the Eustachian catheter with warm iodine, camphor and carbolic.

Result of Treatment.—On testing on February 17, 1926, and again on March 1, 1926, hearing was found to be much improved for all tests; while on March 19, 1926, hearing for conversation and whispered voice was found to be practically normal, and treatment was stopped. Patient has not been tested since but reports further improvement and is able to carry out all examinations in his profession with ease.

Mr. A. R. TWEEDIE asked what was the result of catheterization on the power of hearing before the operation, what was the response to the Gellé test, and if Mr. Barnett tried the effect of plain catheterization afterwards without any "medication" to see if that alone would assist the hearing?

Sir JAMES DUNDAS-GRANT congratulated Mr. Barnett on having been able to induce his patients to undergo thorough treatment. Most otologists knew definitely what was best for patients, but it was often difficult to induce patients to accept the treatment which was best for them.

Mr. H. BANKS-DAVIS asked what Mr. Barnett meant by the term "submucous fracture"; did it mean fracture of the bone after elevating or stripping the mucous membrane?

Mr. A. D. SHARP said he considered that the good results in these cases were mainly due to the submucous resection of the septum, the tonsil operation, and the treatment of defect in the turbinals. He did not follow the rationale of ionization over the mastoid. In his opinion thorough nasal treatment, in such cases, was to be commended and not condemned.

Mr. R. SCOTT STEVENSON said the encouraging point was that Mr. Barnett did make a determined effort to do such patients good. Many otologists, when they met with a case of chronic otitis media or otosclerosis, were apt to fold their arms and say they could do nothing for the patient. Even if simple catheterization was carried out, to mention no other treatment, and the case was persevered with, some improvement often ensued in chronic otitis media. Sometimes it was found that one's patients had not previously had the catheter properly passed into the tube. He criticized the so-called "honest" attitude which led aurists to tell such patients that nothing could be done for them.

Dr. J. S. FRASER said he took a line which was opposed to that of Mr. Scott Stevenson; he was of opinion that many useless operations were performed on patients who were suffering from otosclerosis. The name "chronic catarrhal otitis media" was applied to these cases, but most of them were cases of otosclerosis. He was speaking especially of cases having drumheads which were normal in curvature but were slightly opaque. French women had facial skin which was somewhat more opaque than the facial skin of British women, who consequently showed more pink in the cheeks than their French sisters. Similar differences applied to individual tympanic membranes; one was more opaque than another, yet both were normal. Such operations as enucleation of tonsils, submucous resection of the septum and turbinotomy, were frequently performed on these patients, without making them any better. Sometimes, in cases in which only one ear was affected, the patient was told that the septum should be resected to prevent the deafness spreading to the "other" ear. He (Dr. Fraser) did not think this was fair to the patient. Tonsils might require enucleation in a case of otosclerosis, but only because of tonsil disease—not with any hope of improving the hearing. If the drumhead was normal in curvature, even if opaque, no good could be done by operation on the nose. Otosclerosis was a deeper-seated disease than could be cured or even improved by submucous resection and the other procedures mentioned.

Dr. DAN MCKENZIE (President) said that as one became older one found more and more cases which had been operated upon for dry middle-ear deafness, but the results, in his experience, were disappointing; the deafness continued to increase, whatever was done. Yet, despite that finding and despite what Dr. Fraser had just said, medicine was an experimental science, and not until the experience of many men over a long period had been accumulated could one say how much could or could not be done for these cases. Each one's experience

gave him a particular point of view, and he (the President) held that people who were operating for the conditions under discussion were at the present moment justified. Even if their experiment proved finally that on the whole success was not to be obtained from surgical measures, they were nevertheless doing good work in making the attempt, and there might be a percentage of cases which would benefit.

Mr. NORMAN BARNETT (in reply) said that he always operated, when operation was necessary, before beginning treatment of the middle ear. The operations were only performed when they were demanded on general surgical principles, although he believed the presence of septic tonsils and deflected septum were common predisposing causes of middle-ear deafness. Eustachian catheterization with inflation by air without medicated vapour was of no use, except for a very temporary period.

Submucous fracture of the turbinates was performed by a special instrument which had been made for him. This necessitated no stripping of mucous membrane; the instrument was passed between the turbinates and the septum into the nasopharynx, and gentle pressure exercised when the bone fractured, one part overlapping the other and becoming fixed in that position: it was a preferable alternative to removing the posterior ends of the turbinate bones.

In Case III, as in all of the cases, "history" meant the account given by the patient of his disability, and not his own observations; while "examination" referred to his (Mr. N. Barnett's) personal observation of the case when first seen. "Completely deaf" referred to the patient's statement; "practically normal" referred to his (the speaker's) final examination. In the account of the operation "turbinates" should read "tonsils."

The three cases shown were examples of one cured, one approaching cure, and one better, though still very deaf. They were all cases of progressive middle-ear deafness, and he considered the results were due to suitable treatment having been carried out. The patients in these cases had been not slightly deaf, but very deaf indeed. One patient, a physician, could tell of his inability to perform his examinations, and that now his hearing was practically normal. Another case was that of a schoolmaster who was dismissed by the Board of Education but reinstated after treatment and was carrying on satisfactorily. There were several of these schoolmaster cases. Another case amongst those shown was that of a police inspector who had to have his name removed from the "short list" for a chief constableship because he was unable to hear, and he would probably have had to leave the Force. He was now carrying on normally and his name could be reinstated on the list.

He (Mr. Barnett) divided the patients into three classes: (1) those whom he never saw again and whose hearing as reported to him continued to be satisfactory; (2) those who came at intervals of three or four years for "tuning up"; (3) those who wished to come oftener but were found not to have gone back very far.

Acute Lateral Sinus Thrombosis Following Chronic Otorrhœa.

By J. A. GIBB, M.D.

A. S., AGED 13.

History.—Otorrhœa of some years' duration.

August 23, 1926.—Admitted for acute mastoid radical operation. Lateral sinus opened and plugged. The sinus was thrombosed.

August 25, 1926.—Rigor 3.30 p.m. Temperature 104° F. Internal jugular ligated, tied above facial vein and severed. Lateral sinus further exposed by trephining; septic clot scraped away; clot extended to the neighbourhood of torcular.

August 31, 1926.—Slight rigor 3.15 a.m. 20 c.c. antistreptococcus serum repeated every twelve hours for three days.

September 15, 1926.—Delayed serum rash with swelling of knee-joints: calcium chloride and iron given: condition relieved.

September 22, 1926.—Tonsils and adenoids removed.

October 2, 1926.—Discharged.

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Meningeal Symptoms Developing, Disappearing and Reappearing. Cerebellar Abscess possibly Leaking into Meninges.

By E. Broughton Barnes, F.R.C.S.Ed.

D. J., FEMALE, aged 14. Sent to hospital on October 12, 1926, as a case of meningitis.

History of pain in the left ear three weeks, discharge for four days. Headache forty-eight hours. Bowels not open for four days. Vomited twice previously. No rigors. In letter received subsequently the doctor stated that his diagnosis was based on the headache, vomiting, positive Kernig and general facial expression.

On examination, however, Kernig was negative, Babinski negative. There was no head retraction, the child was not drowsy but appeared quite cheerful and mentally normal. There was a considerable amount of œdema of the tympanum. There was no mastoid tenderness or œdema of the posterior bony meatal wall. Temperature 98·8° F.; pulse 72. Diagnosis of acute otitis media was made.

Forty-eight hours later the child became drowsy during the morning and about mid-day developed a meningeal cry. There was slight retraction of the head. Temperature was 98·4° F.; pulse 80. Operation, October 14: Superficial mastoid cells appeared to be normal, but there was a small deep perisinus abscess and the sinus wall was exuding pus with each heart-beat. The sinus was opened. It was not possible to get below the thrombosis from the mastoid. The internal jugular was tied and a portion excised at the level of the first tracheal ring. The pulse was imperceptible at the end of the operation. Returned to bed. The patient recovered rapidly from the operation, had no further meningeal or intracranial symptoms for a week. There was no nystagmus and no atonia or dysdiadokokinesis. At 3.30 a.m. on the 22nd the patient suddenly started a meningeal cry, her pulse, which had been running between 80 and 100, suddenly fell to 52. She had a deviation of the eyes to the right with occasional flicks of cerebral correction, and rapidly became unconscious. She had a marked positive Kernig. Lumbar puncture: 5 c.c. of clear fluid was withdrawn under very slightly increased pressure. Two hours later, operation through the mastoid incision: cerebellum was explored through the sinus wall, which was adherent to the cerebellum. Cerebellum was obviously compressed, and at a depth of half an inch an extremely stout abscess wall was found. On opening the abscess a slightly turbid fluid in considerable quantity was followed by pus which appeared to be *in loculi* and came in rushes as the forceps were moved in the cavity. The pus was followed by cerebro-spinal fluid. The abscess wall was so stout that a thick rubber drainage tube was immediately collapsed by it. A T-incision had to be made in the abscess wall to drain it.

Twelve hours later the patient was unconscious and apparently dying. The pulse was 136 and extremely weak. At half-past six she suddenly sat up and asked for an orange. She had then lost all nystagmus and deviation, but her left side was notably atonic. Lumbar puncture fluid containing short chain streptococci and Gram-negative bacilli. The streptococci grew on culture, the bacilli did not. Pus from the abscess contained the same organisms. Forty-eight hours later, October 28, lumbar puncture fluid under slight pressure contained streptococci which did not grow on culture. Cerebro-spinal fluid drained through the tube in the abscess for three days.

October 29.—Lumbar puncture: slightly increased pressure, no organisms in the fluid. The dysdiadokokinesis and atony persisted and became slightly more marked early in November. On November 16, 1927 radical operation was completed

and a skin graft placed which did not take well. Recovery from this point was uneventful. The child did not complain of headache after the end of October. Her tonsils and adenoids were removed on December 21.

There have since been troublesome granulations from the anterior part of the radical cavity.

I think that the cerebellar abscess was leaking into the meninges at intervals.

Intracranial Complication of Aural Suppuration.

By E. BROUGHTON BARNES, F.R.C.S.Ed.

M. W., GIRL, aged 13. Sent to hospital as an emergency case, April 5, 1927, with a diagnosis of cerebral abscess.

Previous History.—Scarlet fever when aged 10; right ear had discharged since; fourteen days previously discharge had ceased; some pain in neck; three attacks of "biliousness," last one three weeks previously; vomiting and ? giddiness; nine days previously, severe headache "all over the head"; one week previously, shivering attack.

Condition on Admission.—No headache, bright and intelligent, does not look ill. Pulse 80. Temperature 98·4° F. Visual fields normal. All reflexes normal. No tenderness or œdema of mastoid. No œdema of posterior bony wall or tympanum.

Twenty-four hours later, after a good night and day, patient suddenly became drowsy, vomited. Pulse fell to 62.

Radical Mastoid Operation.—Sclerotic mastoid; large cholesteatoma cavity which had exposed the dura of the middle fossa and the lateral sinus, both apparently healthy and without granulations. Fistula of bony external semicircular canal. Patient bright and cheerful next day, and free from headache. No nystagmus. Weber to the right.

Patient apparently progressed very well until April 11, 1927, when, at 4 a.m., she complained of severe headache and nausea and looked ill and anxious. Pulse 68. No retraction of head. Kernig to 45 degrees only, both legs. Sinus was pulsating well. Dura of middle fossa appeared slightly more tense.

Operation.—Incision extended, and dura of middle and posterior fossæ further exposed. Dura appeared normal in both areas, except that the tension of the middle fossa was high; pulsation was good. Exploration of temporo-sphenoidal lobe: Large area of lobe above tegmen tympani, apparently about 1½ in. square, pulpy, and having small beads of pus at intervals. No abscess wall. Not adherent to dura. Multiple incisions made in dura and temporo-sphenoidal lobe widely explored. No pus except the diffuse drops in brain substance. The pulpy brain herniated through the incisions. No tube was inserted.

Lumbar Puncture.—50 c.c. turbid fluid withdrawn, crowded with streptococci and polymorphonuclear leucocytes. Streptococci did *not* grow in culture.

After Treatment.—Incisions kept open with forceps for five days at daily dressings.

Patient has had an uneventful convalescence. No further lumbar puncture has been made. Wound was closed on May 17, 1927.

Discussion.—Dr. DAN MCKENZIE (President) said that last year he had shown specimens of brain and meninges from a similar case. Meningitis symptoms appeared and disappeared three or four times, the child being ill three months. The reason of the death was that there was a double abscess, a deeper satellite abscess had escaped notice. These cases showed that the meninges had great power of resistance against infection. In his own case the arachnoid space was occupied by tough fibrous tissue as a result of the sepsis.

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Dr. J. S. FRASER said Mr. Barnes was to be congratulated on his results. What would be of value would be for some Members of the Section to give the results of *all* their cases of meningitis. These two cases and their results gave a wholly erroneous impression of the usual percentage of cures in such conditions.

Mr. J. A. GIBB said he had a case of the kind in a man aged 25 who had chronic otorrhœa with a fulminant mastoid, thrombosis of the lateral sinus and abscess of the temporo-sphenoidal lobe. He was treated and made a complete recovery, the wound healed, and he (the speaker) went away for a holiday. Three days before his return the man had a fit and died. There was no post-mortem examination.

Mr. BROUGHTON BARNES (in reply) promised to write and send in a list of his cases, procedure and results.

The two cases shown were actually consecutive, but he did not claim anything like a hundred per cent. successes. His cases were too few at present for statistical purposes.

Section of Psychiatry.

President—Dr. W. Aldren Turner, C.B.

The Feeling of Inferiority and the Striving for Recognition.

By Alfred Adler.

(Translated by Walter Béran Wolfe, M.D.)

I.—The Situation in Early Childhood.

We recognize the fact that children who have been treated as step-children by Nature have an entirely different attitude toward life and their fellow creatures from that of those to whom the joys of existence were vouchsafed at an early age. It may be stated as a fundamental law that children who come into the world with organic defects become involved at an early age in a struggle with the facts of existence which results only too soon in the suppression of their social feeling. Such children are continually preoccupied with themselves and the impression they make upon others, instead of interesting themselves in an adjustment to their fellows. What holds good for organic defects is equally valid in the case of any social or economic disability which manifests itself as an additional burden capable of producing a hostile attitude toward the environment. The deciding trend becomes determined at an early age, and such children have an intuition, even at the age of two, that they are somehow not as adequately equipped for the struggle as their playmates, and have a sense that they cannot trust themselves in the common games and pastimes. As a result of past privations they have acquired a feeling of being neglected, which expresses itself in their tendency to hold themselves in an attitude of anxious expectation. It is necessary to remember that every child occupies an inferior position in life, and were it not for a certain quantum of social feeling on the part of his environment would be incapable of independent existence. One can realize that, at the commencement of every life, there must be a more or less deep feeling of inferiority. This arises in the weakness and helplessness of children who are at all conscious of their inability to cope single-handed with the problems of existence. This feeling of inferiority is the driving force, the starting point, from which every childish striving originates. It determines how, and whether, this individual child acquires peace and security in life, it determines the very goal of his existence, and prepares the path along which this goal may be reached. The basis of his educability lies in this attitude, this peculiar situation in which every child finds himself, a situation very closely bound up with his organic potentialities. This educability, general as the feeling of inferiority is, may be destroyed by two factors: (1) An exaggerated, intensified, unresolved feeling of inferiority, and (2) a goal demanding not only security, peace and social equilibrium, but power over his environment, i.e., dominance over his fellows. Children who have such a goal are easily recognized and they become "problem" children, (1) because they interpret every experience as a defeat, and (2) because they always

consider themselves neglected and discriminated against by both Nature and mankind. One need only consider these factors to see how readily a crooked, inadequate, error-ridden development may occur in the life of a child.

Every child runs the danger of such a development, because he finds himself, at some time or another, in a situation which is precarious.

A child finding himself in an environment of adults is predisposed to consider himself weak, small and incapable of living alone, and in such a situation is quite unable to trust himself to accomplish little tasks, which we would think him capable of performing without making mistakes, and exhibiting clumsiness of action. At this point most of our errors of education commence, and by our demanding more than the child can accomplish, the idea of his own helplessness is thrust upon him. Some children are consciously made to feel their smallness and helplessness. Then, again, there are children who are regarded as merely animated dolls, and those who are considered valuable property that must be carefully watched. Lastly, there are those who are made to feel that they are useless human freight. A combination of these attitudes on the part of parents and adults, generally leads a child to believe that he can accomplish only two things, the giving of pleasure or displeasure to his elders. The type of inferiority feeling which is produced by such an attitude on the part of the parents may be further intensified by other peculiar characteristics of our civilization. The habit of not taking children seriously falls into this category, and a child gets the impression that he is a nobody and without rights, that he is to be seen, not heard, must be courteous and quiet, etc. There are numerous children who grow up in the constant dread of being laughed at. This laughing at children is well-nigh criminal; it retains its effect upon the soul of the child, and upon his reaching adult life is transferred into habits and actions. One often recognizes an adult who was continually laughed at by his parents when a child; such individuals cannot rid themselves of the fear of being made ridiculous even on reaching maturity. Another aspect of this matter of not taking children seriously is the custom of telling children palpable lies, with the result that the child begins to doubt, not only his immediate environment, but also to question the seriousness and reality of life.

Cases have been recorded of children who laughed continually at school, seemingly without reason, who, when questioned, admitted that they thought school was a joke of their parents, not worth taking seriously!

II.—Compensating for the Feeling of Inferiority; the Striving for Recognition and Superiority.

It is the feeling of inferiority, of inadequacy, of insecurity which determines the goal of an individual's existence. The tendency to push into prominence, to attract the attention of parents, makes itself felt in the first days of life. These are the first indications of the awakening striving for recognition which develops itself under the concomitant influence of the feeling of inferiority, and purposes a goal in which the individual is seemingly dominating his surroundings.

The degree and quality of the social feeling also helps to determine the goal of dominance. We cannot judge any individual, whether he be child or adult, without drawing a comparison between his goal of dominance and the quantum of his social feeling. His goal is so determined that its achievement promises the possibility of either a sentiment of superiority, or, at least, an elevation of the personality to such a degree that life seems worth living. It is this goal which gives value to our sensations, which links and coördinates our sentiments, which shapes our imagination, guides our creative powers and determines what we shall remember and what we must forget. We can realize how relative is the value of all our sensations, sentiments,

affects, imagination, when we appreciate the fact that not even our sensations are absolute quantities. All are influenced by the striving for a definite goal, so that our very perceptions are prejudiced and always chosen, so to speak, with a secret hint at the final end and toward which the personality is striving.

We orientate ourselves according to a fixed point which we have artificially created, and which does not exist in reality. This assumption, necessary because of the inadequacy of our psychic mode of expression, is very similar to the fictions which are of use in other sciences, such as dividing the earth with meridians; these do not actually exist, but are useful assumptions. In the case of all psychic fictions we have to postulate the following: We assume a fixed point even though close observation forces us to admit that it does not exist. The purpose of this assumption is simply to orientate ourselves in the chaos of existence, so that we can come to some apperception of relative values. The advantage is that we can classify every sensation and every sentiment according to this fixed point, once we have assumed it.

Individual psychology, therefore, creates for itself a heuristic system and method; it regards human behaviour, and endeavours to understand it, as though it were possible to produce a final state of affairs out of a set of inherited potentialities which are all tending to strive for a definite goal. Our experience, however, has shown us that the assumption of a striving for a goal is more than simply a convenient fiction. It has proved itself to be largely coincident with the actual facts in its fundamentals, whether these facts are to be found in the conscious or unconscious life. The striving for a goal, the purposiveness of the psychic life, is not only our philosophic attitude, but actually a fundamental fact.

When we question how we can, in a case of the child, best meet and answer the question of the abolition of that striving for power which is a prominent evil of our civilization, we are faced with the difficulty of making ourselves understood. For this striving tendency shows itself in the life of a child long before the time when it is possible to begin making attempts at improvement and clarification. Nevertheless, our living with the child, when such tendencies first occur, does afford us the opportunity of developing his social feeling so that his striving for personal power becomes a negligible factor.

A further difficulty lies in the fact that even children do not express their striving for power openly, but hide it under the guise of gentleness. They conceal their aspirations as if behind a veil, and so modestly expect to escape disclosure in this way. Their uninhibited striving for power, seeking for support, is capable of producing degeneration in their psychic development, with the result that, in their exaggerated striving for security and might, courage becomes changed to impudence, obedience into cowardice, gentleness degenerates into a subtle treachery, and the final result is that every natural feeling or expression carries with it a hypocritical afterthought the final end of which is the subjugation of the environment.

Conscious education works upon the child with the conscious, or unconscious, desire to remove him from his insecurity, to school him in the technique of life, give him a knowledge and educated understanding, and furnisn him with a social feeling for his fellows. All these measures, from whatever source they come, are to be understood as attempts to give the growing child new ways of ridding himself of his insecurity and his feeling of inferiority. What happens in the soul of the child during this process we must judge by his character traits which are the mirror of the activity in his soul.

The importance of the feeling of insecurity and inferiority depends largely upon the interpretation given to it by the child; the objective degree of inferiority is important, of course, and makes itself felt.

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One must not expect a child to possess the correct estimation of himself in this situation, any more than one expects adults to possess it. Here the difficulties grow apace, as one child will grow up in a situation so complicated that errors concerning the degree of his inferiority are absolutely unavoidable, while another child will be better able to interpret his situation. But taken as a whole the interpretation which the child has of his feeling of inferiority varies from day to day until it finally becomes consolidated and expresses itself as a definite self-estimation. According to this crystallized self-estimation, the compensatory trends which the child creates to guide him out of his inferiority will be directed toward this or the other goal.

The mechanism of the striving for compensation, by means of which the soul seeks to neutralize the torturing feeling of inferiority, has its analogy in the organic world. It is a well-known fact that those organs of our bodies which are essential for life produce an overgrowth and overfunction when, through damage to their normal state, their productivity is lessened. Thus, in difficulties of circulation, the heart enlarges and becomes more powerful, seeming to draw its new strength from the whole body, until it reaches a stage in which it has become more powerful than a normal heart. Similarly so, does the soul under pressure of the feeling of inferiority, of the torturing thought that the individual is small and helpless, attempt with all its might to become master over this " inferiority complex."

Where the feeling of inferiority is highly intensified, to the degree at which the child believes that he will never be able to compensate for his weakness, the danger arises that in his striving for compensation he will be satisfied not with a simple balance of power, but will strive for an extra-compensation and will aim at over-balancing the scales.

This striving for power and dominance may become exaggerated and intensified to a degree that will entitle it to be called pathological. The ordinary relationships of life will never satisfy such children and, varying with their goal, their movements will come to assume a certain grandiose gesture. They seek to secure their position in life with extraordinary efforts, with greater haste and impatience, with more intense impulses, and without consideration for anyone else. Owing to these exaggerated movements toward their exaggerated goal of dominance, such children become more noticeable, and their attacks on the lives of others necessitate defensive measures against their activities. They are against the world, and the world is against them, and this antagonism does not necessarily occur in a bad sense of the word. Such a child may express his striving for power in a manner which does not come into conflict with society for a long time, and his ambition may be considered as no abnormal characteristic. If, however, we carefully investigate their accomplishments and achievements, we shall find that for the most part society at large does not benefit from their triumphs, because their ambition is a social one and will always put them in the path of other human beings as disturbing elements. Gradually other characteristics will appear which will evince a more anti-social colour, if we consider an individual in his full relationship to his fellow-men. In the forefront of these manifestations we find pride, vanity, and the striving to conquer everyone regardless of method, and this striving may manifest itself subtly in the relative exaltation of the individual, through the degradation of all those with whom he comes in contact; the important thing being only the distance which separates him from his fellows. Such an attitude is not only uncomfortable for his neighbours, but it is also very uncomfortable for the individual, because it brings him into continual contact with the dark side of life and prevents him from experiencing any happiness.

By means of the exaggerated straining for power with which these children

endeavour to ensure themselves a place over their environment, they soon put themselves in a position of resistance to the ordinary tasks and duties of everyday life. Compare such a "power-hungry" individual with the ideal social being, and one can, after some little experience, specify, so to speak, his social index, that is, the degree to which he has removed himself from his fellow-man.

The student of human nature, in observing one of these individuals, will immediately recognize that some difficulties in the evolution of the psychic life must have occurred here, even as his eyes are open to any physical deficiencies and inferiorities. If we keep this fact in mind we shall do no damage to the individual's social feeling, seeing that we ourselves have developed an adequate social feeling, and with this attitude only we can help the individual. Bearing this in mind, we must not blame the bearer of a psychical defect or a disagreeable characteristic for his attitude of indignation. Indeed, we must admit his right to be indignant to the last limits, and we must be conscious that we participate in the common blame for his situation. We share the blame because we, too, have taken part in the inadequate precautions against the social misery which has produced it. If we abide by this standpoint we may effect improvement, and only thus can we approach such an individual, not as a degraded, worthless outcast, but as a fellow human being. Having this attitude toward our patient we must surround him with an atmosphere in which he will find that there are possibilities for feeling himself equal to every other human being in his environment. We have only to remind ourselves how unpleasant may be to us the sight of an individual whose organic or bodily inferiorities are externally visible, to judge the amount of education which we ourselves need in order to arrive at an absolutely just evaluation of our social feeling, and to judge also how much our civilization owes to such an individual. It is taken for granted that those individuals who come into the world with organic defects feel existence to be a burden from their earliest days—a suffering which most of us are spared. They thus find themselves in a position of pessimism as regards the whole structure of existence. Children who have less noticeable organic defects, but in whom the feeling of inferiority has become intensified, from whatever cause, right or wrong, find themselves in a very similar situation. Such a feeling of inferiority may be so artificially intensified, as, for instance, by over-pressure of education during the critical period, that the result is exactly the same as though the child came into the world greatly crippled. They never rid themselves of the thorn which has pierced their side in the early days of their existence, and the coldness which they have experienced prevents them from approaching their fellow-beings, with the result that they believe themselves to be in a world devoid of love and affection and with which they have no point of contact.

For example: A patient became specially noticeable because he was continually telling us about his great sense of duty and the importance of all his actions. He lived with his wife in the worst possible relationship, both he and she being individuals who measured to a hair-breadth the value of any event as a means toward the subjugation of the other partner. The result was seen in continual wrangling, reproaches, and insults, in the course of which the two had become entirely estranged from one another. This individual certainly retained some small portion of his social feeling for his fellow-men, but even this was suppressed by his striving for superiority, at least so far as his wife and friends were concerned.

We learned the following facts from the story of his life. Up to his seventeenth year he was practically undeveloped physically. His voice was still the voice of a young boy, he had no hair on his face or body, and he was one of the smallest of the boys with whom he associated. Now at thirty-six years of age his external appearance is normal and is entirely masculine; Nature has seemingly caught up with

herself and completed everything which she had hardly begun to fashion when he was seventeen. But for eight years he suffered from this failure of development, and he had no ground for hoping that Nature would ever compensate for the fallow time. During this entire period he was tortured with the thought that he must always remain a "child," and already the beginnings of his present characteristics could be noted. He acted as though he were very important and as if every action had the utmost weight. Every movement served the purpose of bringing him into the centre of attention. In the course of time he acquired those characteristics which we see in him to-day. During married life he was continually occupied with impressing upon his wife the fact that he was really bigger and more important than she thought, and she was continually busied with showing him that his assertions concerning his value were untrue! Under these circumstances their marriage (both of the partners having already shown signs of disruption during their engagement) could hardly be expected to develop favourably, and the end was a social cataclysm. At this point, however, the patient came to the physician, since the dissolution of his marriage had served only to accentuate the dilapidation of his already battered self-esteem. In order to effect a cure, the first thing to be learnt from the physician was how to know human nature, and to estimate the extent of the error he had made in life. And this error, this wrong evaluation of his inferiority, had coloured his entire life up to the time of his coming for treatment.

Section of Surgery.

President—Mr. V. Warren Low, C.B., F.R.C.S.

DISCUSSION ON THE TREATMENT OF CLEFT PALATE BY OPERATION.

Sir James Berry, F.R.C.S.

Introductory Remarks.

Sixteen years have elapsed since the operative treatment of cleft palate was the subject of a debate by the Section of Surgery of the Royal Society of Medicine.[1] It has been thought by many of us that the time has now come for another debate on the same subject. It is hoped that most of those surgeons of this country who are especially interested in this branch of surgery and have had much practical experience of it, will respond to the invitation of the Section to relate their experiences and their views and to show some of the patients upon whom they have operated.

At the last debate the main point at issue was the relative value of the turnover flap operation performed in very early infancy, as compared with the operation of lateral approximation of the edges of the cleft. With the question of the relative merits of these two classes of operation is closely associated that of the age at which in the best interests of the patient the operation should be performed. The main, indeed almost the only, object of operating upon a cleft of the palate is to enable the patient to speak intelligibly. The view formerly held by some that the operation is to be regarded as a "life saving" one is now, I venture to think, an exploded one, maintained at the present day by hardly any surgeons who have had much practical experience of cleft palate surgery. That a cleft palate infant, if neglected, may die of inanition is true enough, for it can but seldom be fed at the breast. But a cleft of the palate does not prevent an infant from swallowing. The proper way to deal, therefore, with a newly born infant afflicted with a cleft palate is, of course, to feed it carefully, *not* to perform upon it at once an extensive operation with its attendant loss of blood and sepsis. Any intelligent mother or nurse can easily be taught the proper method of administering milk, preferably the mother's milk, given by means of a teaspoon, to a newly-born infant with a cleft palate.

There is a certain small proportion of clefts of the palate which are very narrow and limited to the soft palate, or part of it, which could doubtless be closed within the first few weeks or months of infancy by the operation of lateral approximation, were it thought desirable to do so. But in the great majority of single or double clefts of the palate, it is impossible at this very early stage to perform a lateral approximation operation with any reasonable chance of success, owing to the width of the cleft and the lowness of the palatine arch, in which the alveoli are but slightly developed.

It is, of course, well known that a complete cleft of the palate, wide at birth, becomes gradually narrower as the child grows. To a less extent the same process may be observed in clefts which involve only a portion of the hard palate and sometimes even in cases in which the soft palate alone is involved. This gradual narrowing of a complete cleft takes place more rapidly when the accompanying hare-lip is closed, as it should be, within the first few months of life.

[1] *Proc. Roy. Soc. Med.*, 1911, iv (Sect. Surg.), 169-198.

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If any operation for an extensive cleft of the palate must be performed in very early infancy, the operation must almost necessarily be some form of turnover flap operation, unless the operation of forcible approximation of the jaw-bones by wires, or other mechanical means, be adopted. To the latter operation there are many serious objections which I need not here discuss, as in this country, at least, it is now, I believe, but rarely practised.

As regards the age at which a cleft palate operation should be performed, I would say that it should be done at the earliest age at which the experience of the operator leads him to think that by a lateral approximation operation he will have a reasonable chance of closing the cleft. This will seldom be in the first year of life, but generally at some time between eighteen months and three, or possibly four, years, of age. An operator who has had little or no experience of this branch of surgery and who finds himself called upon to do one of these operations will even do well to follow the old-fashioned plan of postponing the operation until a much later period. This is better than running the risk of ruining a child for life by spoiling its palate and possibly leaving it in such a condition that no other surgeon has any chance of remedying the condition. We have all seen examples of this and we shall doubtless all agree that the operations that we have to do upon palates that have already been operated upon without success are more difficult to perform and usually less successful in their ultimate results. The consequent loss of tissue and the presence of scarring are the principal causes of this greater difficulty.

Everyone will admit that there is a certain, although small, advantage to be gained by closing a cleft palate before the child has learnt to speak, if this can be done with a reasonable chance of success.

Since the date of the last debate, I have attempted tentatively, in a certain number of cases, to endeavour to close the cleft at a somewhat earlier age than was formerly my practice. But, on the whole, I have been disappointed with the results of these operations. In a large proportion of cases the immediate result has been that more or less of the hard palate has remained unclosed and a subsequent operation has been necessary. Whether it is desirable to close the soft palate at a relatively early age and to leave the hard palate to be operated upon later, is a point upon which there will doubtless be difference of opinion. Personally, I do not think that there is much to be gained by this two-stage proceeding. The subsequent operation upon the hard palate is generally made more difficult, owing to the presence of scar tissue and the greater difficulty of effecting that separation of the soft tissues from the posterior edge of the bony hard palate which is so essential for the success of a cleft palate operation.

In a really difficult case, such as that of a very wide cleft involving the soft palate and most of the hard palate—the most difficult of all cases—it is often, in my opinion, a good plan to close the cleft of the soft palate first, and to leave the hard palate unclosed for a time rather than to risk failure of the whole line of union by attempting too much. In many of such cases, and in some cases of complete cleft, I often leave the anterior end of the cleft unclosed, rather than endanger the vitality of the flaps by severance of the anterior palatine artery. The small hole thus left will often close spontaneously, or can, without much difficulty, be closed by a small subsequent operation. And here I would express the strong opinion that it is the soft palate that it is most important to close. If after an operation for cleft palate, the hard palate has been closed and the soft left unclosed, but little good has usually been done to the patient. On the other hand, if the soft palate be successfully closed (and this can nearly always be done), and the hard palate left unclosed, the patient has been greatly benefited. The aperture in the hard palate can usually be closed at a later date, or, at the worst, can be fitted eventually with an obturator.

No obturator with which I am familiar can really satisfactorily take the place of a well-restored soft palate.

Turning now to the details of the lateral approximation operation which most of us now employ, there are several points about which there will, I am sure, be much difference of opinion. If I venture to state my own views somewhat dogmatically I do so with the express object of promoting discussion and criticism. I may say that my views are based upon my experience of the 276 cleft palate patients upon whom I have operated in the last thirty-three years (up to the end of 1926).

Before operation, gross defects of teeth and tonsils should be remedied by suitable means. Anything like routine excision of the tonsils or adenoids I believe to be quite unnecessary. Nor do I believe that much benefit is to be obtained by any systematic attempt to render the mouth aseptic by mouth washes or spraying for days before the operation. I never do this. On the other hand the general health of the child must be good, and if the child has nasal catarrh, operation must always be postponed till this has been cured. It is well also to remember that if the general nutrition is poor and the child is thin, the soft tissues of the hard palate will be thin, and the suture of its cut edges much more difficult to perform with success.

Many operators prefer to sit at the head of the patient and to have the head lower than the body. Personally, I do not like this position, as it leads, I think, to undue venous congestion of the veins of the palate and consequently to greater loss of blood. Anything like extensive loss of blood has a most prejudicial effect upon the healing of the wound. I myself always operate standing on the right side of the patient and facing the head. The patient's shoulders are well elevated and the head is thrown well back, so that the blood runs into the upper part of the pharynx and not into the larynx. This position, on the other hand, has the disadvantage of sometimes producing a tendency to faintness which may cause the anæsthetist, if inexperienced, a temporary embarrassment. But there is less hæmorrhage than when the other position is adopted. It is of the utmost importance that the patient should not lose much blood. What kind of gag be employed, or whether no gag at all be used, the tongue being simply drawn forward by forceps or by a thread passed through it, are matters of but little importance. Each operator will use the gag or method with which he is familiar. My own preference is for Smith's gag, but the assistant who holds it must have been well trained to do so.

The various steps of the operation I need not discuss at any length. They have been fully described and illustrated in the book published by Mr. Percy Legg and myself in 1912[1] with which doubtless most of you are familiar. There are two points, however, which I think merit full discussion and one in which I believe my present practice is in advance of that which I have advocated up to the time of my last publication on this branch of surgery.

The first point is the extent to which the soft tissues should be separated from the bony palate. If this be not done thoroughly, even in clefts limited to the soft palate, the soft palate cannot be brought down sufficiently to enable the pared edges to be brought together without tension. If there is tension there will almost certainly be failure of union. Want of attention to this point is, in my opinion, one of the commonest causes of failure in the operation. The separation is best done, I think, from the inside of the cleft. Care, however, should be taken to avoid the neighbourhood of the posterior palatine artery. It is quite easy to cut too freely in this region and thereby not only to endanger the vitality of the separated soft parts but also to cause atrophy and paralysis of the soft palate. I think that in some of my earlier operations, in my anxiety to approximate the edges of the cleft without

[1] "Hare Lip and Cleft Palate," J. and A. Churchill, London.

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undue tension, I have cut the soft parts too freely and left the soft palate anatomically apparently normal, yet flaccid and paralysed. It should not be forgotten that the nasal intonation of cleft palate speech is due to abnormal escape of air through the nose. Normally this escape is prevented not only by the elevation of the soft palate, but also by the lateral approximation of the palato-pharyngeal muscles. Normally these muscles form a kind of sphincter, capable of shutting off the oral pharyngeal cavity from the naso-pharynx. In cleft of the palate this sphincter is incomplete. The aim of the surgeon should be to restore and complete this sphincter, not to damage it unduly.

For the same reason, the lateral cuts which are made by scissors on the nasal surface of the soft palate, in order to bring the latter downwards and towards the middle, should not be too extensive. The levatores palati muscles, if cut at all, should be cut close to the soft palate, and in one place only, not snipped in several places. The palato-pharyngeal muscular fibres will seldom require any division at all, although the mucous membrane covering them may often be cut through with advantage and without doing any harm.

The next point that I think we might discuss with advantage is the question of lateral incisions to relieve tension. How far are they necessary? If used at all, where should they be placed and how long should they be? In the case of wide and difficult clefts they are frequently necessary, on one or both sides. But they should, in my opinion, be as short as possible, seldom more than $\frac{1}{2}$ in. in length, except in the case of secondary operations for large holes in the hard palate. In these latter cases if the incisions are situated wholly in front of the posterior palatine canals and not too near the cleft, they may often be made of considerable length without endangering the vitality of the soft parts, and in this situation, of course, they do not damage the soft palate. But of late years I have been using lateral incisions less and less, replacing them as regards the soft palate by the use of deep tension-stitches and rubber plates after the method recommended by Brophy, although I do not employ the lead plates and silver wire which he has advocated. I began many years ago—before 1912—by using wide-tension stitches of stout fishing gut to the soft palate. Then, as I found these tended to cut out, I put short pieces of rubber tubing on the suture. These tended to cockle and cause ulceration. Then I used curved pieces of rubber cut from large drainage tubes, and finally these were replaced by flat oval discs of sheet rubber about 2 mm. thick, threaded on stout fishing gut. These I have been using for many years up to date and find them quite satisfactory in bad cases. It is not necessary to use them in all cases, but I use them in all except easy cases. These plate-bearing sutures should be passed through the soft palate as far away from the edges of the cleft as possible. Care must be taken to place the plates exactly opposite each other. It has been objected against them that they increase the liability to sepsis, but I have not found this to be the case to any serious extent, provided that they are removed sufficiently early, generally on the fifth day. By this time the cut edges of the soft palate should be sufficiently united to stand any strain that may be put upon the line of union by the palatine muscles. If left longer than five or six days they are apt to cause ulceration. They are of course not to be employed on the much thinner tissues of the hard palate where they would be perfectly useless.

Of the very long lateral incisions recommended by some, I have no personal experience. Frankly I am afraid to use them as I cannot help thinking that they must damage the palatine and pharyngeal muscles too much. But on this point I look for further light from advocates of their use. I have no doubt that these incisions facilitate the mere closure of the cleft.

There are, doubtless, numerous other points of detail that will be raised in the course of this discussion.

As regards treatment of the case after operation, I would merely express my opinion that the less that is done the better. The patient is fed on nothing but milk or milk and water, for at least a week. No sort of washing or spraying or wiping of the mouth is permitted, except that on each occasion after feeding, the child is given a drink of warm water. The patient is kept out of doors as far as possible if the season and weather permit. Even rain does not prevent the child being taken out in a perambulator. On the fourth day I usually inspect the palate for the first time and decide when the plates should be removed.

I am of opinion that more harm is usually done by injudicious attempts at active after-treatment than by leaving the patient mainly to the care of a well-trained nurse. If the operation has been carefully and properly done, with gentleness, avoiding excessive bruising of tissues and much loss of blood, it is seldom that a complete failure occurs.

With regard to the speech results of my lateral approximation operations, I have on previous occasions[1] published full details of two long series of cleft palate operations (on sixty-seven and eighty-one patients), noting in every case the ultimate speech result when it was possible to do so. To these have been added a few other cases, bringing the total number up to 154, published in full detail in the Appendix II, of the book already mentioned. On several occasions before this Society and elsewhere I have exhibited many patients and demonstrated the speech results. Fifteen were shown on the occasion of the last debate at the Royal Society of Medicine, and I propose to show some more this afternoon. But now that I have given up most of my hospital work I find it much more difficult to follow up my cases and to tabulate my results as I have done before. I am glad to see that Mr. Rayner, of Manchester, has also recently[2] published in tabular form his results in a consecutive series of 125 cases, with admirable comments upon them. It is to be hoped that others will now follow the examples that we have set them.

Mr. G. GREY TURNER (Newcastle-upon-Tyne).

My part in this discussion can only be to place before you my personal experience of the management of cleft palate. The Council of the Section have laid down certain points on which information would be most valuable, and I shall first devote myself to those heads.

The type of cleft has a bearing on several of the problems involved. In my series the proportion of the different varieties is shown in the following diagram (fig. 1). At the outset I would briefly discuss the responsibility of advising operation in cases of cleft palate. It is not unnatural that considerable attention should have been focused on the many unsuccessful results that are often seen after attempts to cure this deformity by operation, and on the very good speech that is sometimes attained by those who have never been operated upon at all; also on the great benefit which may be derived from the use of obturators made by dentists. The few adults who have not been operated upon and who speak passably, or sometimes even as well as they would have done if an operation had been most successful, have probably escaped an operation because their speech has always been good, and this observation is in keeping with that made in those cases where speech is good after a bad anatomical result. Had these patients spoken as badly as most sufferers from cleft palate, then an operation would have been urged in infancy. Quite apart from the question of speech, a patient who has a united palate is more comfortable than otherwise; food does not enter the nose, there is less liability to colds, and the victim is not distressed by the cumbersome apparatus which has often to be worn when the palate is not closed by operation.

When the services of a surgeon who has been properly trained in cleft-palate work are available, I consider it a duty to advise an attempt at operative closure. In trained hands the soft palate can nearly always be successfully repaired, though an anterior defect may have to

[1] *Brit. Med. Journ.*, 1905 (ii), 853; and 1911 (ii), 1092.

[2] *Lancet*, April 18, 1925.

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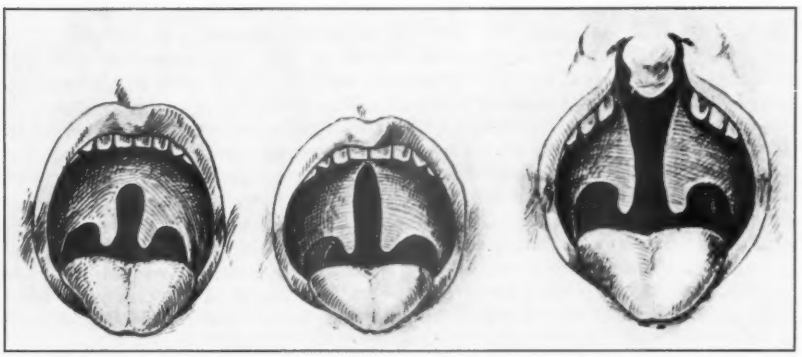
be left to be dealt with by the dentist. There is this further point, that if a child is not operated upon in infancy, it may reasonably feel, when it arrives at years of discernment, that an attempt ought to have been made.

The standard of speech must also be taken into consideration, for parents are often content, or at least not greatly concerned, because they and their families can easily understand what the sufferer says. Of course, the real criterion of the efficiency of the speech is that *strangers* should easily be able to understand, and nothing less than this should be allowed to suffice.

In my opinion not enough has been said about the many cases in which an ideal result has been obtained by surgical means, and I take it that it is part of the object of this discussion once again to bring the actual results before the profession and to demonstrate that satisfactory results, both anatomical and functional, may often be attained.



a *b* *c*

FIG. 1.—TYPE OF CLEFT IN 155 CASES.

(*a*) From middle of hard palate or soft palate alone, 78 cases.
(*b*) Right up to back of alveolus, 19 cases.
(*c*) Complete clefts, 58 cases.

The following are some statistics of the simplest sort:—

Total number of cases		...	...	155
,,	,,	operations	...	...	195
,,	,,	deaths	...	...	4

Causes of Death.—Hæmorrhage, scarlet fever (2), under anæsthetic.

Average age (excluding cases over 12) at time of operation, 4 years and 4 months. Average age of twenty-four cases over 12 years of age, 18 years 9 months.

In twenty-four cases one or more operations had been carried out before the patient came under my care, so that the total number of operations to which the 155 patients submitted was 219.

I ought to explain that such procedures as the preliminary extraction of teeth or the removal of tonsils and adenoids, or subsequent interference for the removal of sutures, cauterization of holes, etc., have not been counted. The figures, therefore, give an indication as to how often it is necessary to carry out re-suture of some part of the palate, or to interfere for the closure of holes, or to repeat an operation which has perhaps been a complete failure so far as the holding of the sutures is concerned. Several of my patients still require some such interference so that the figures show the enormous amount of work that has to be expended on these cases before the results can be "finalized."

Deaths.

The statistics include all the cases on which I have operated, and reflect my early experience. But except for such regard for bleeding as would prevent the loss of a patient from that cause, I cannot claim that a more mature experience has taught me how to avoid occasional calamities. The death under anæsthesia was proved post-mortem to be due to an enlarged thymus. The patient was a healthy but rather plump child of 15 months, and an easy case to operate upon. Ether was skilfully administered by a careful anæsthetist of large experience in cleft palate work. The deaths from scarlet fever were in patients aged three years and nine months, and four years, respectively. The disease developed on the third and fifth days and the children died in the fever hospital. In both the mouth became infected and the palate sloughed.

Age at Which Operation Should be Carried Out.

The time to do the operation is not only a question of the age, but of the health of the patient. Obviously no case should be touched in the presence of a local or general infection; but I have more in mind the general nutrition, especially with regard to anæmia. Before it is decided to operate, the child should be thriving, should look well and should not be lethargic. I am always suspicious of the resisting powers of the light-haired, angelic child; some of the

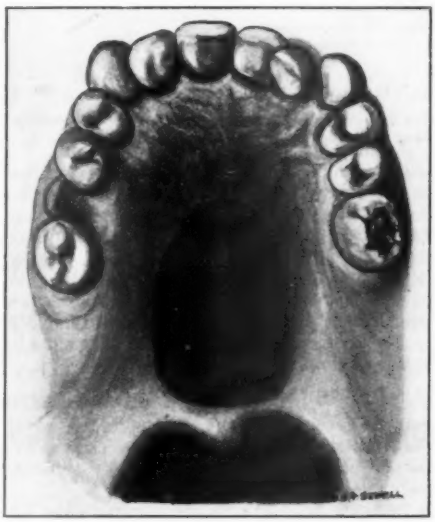


Fig. 2.—"Munn," age 11. Operation at 4 years. Palate sloughed and boy nearly died. Congenital syphilis.

best results as far as healing is concerned have been the troublesome children! Lack of appetite is a bad sign and those who take nourishment with avidity usually do best. I have been much impressed with the importance of this question of the general health, and I have seen many striking instances of its bearing on the result.

In the case of F. P., aged two years and three months, the conditions appeared to be very favourable except that the child was light-haired and anæmic. The complete operation was carried out in March and was easy and apparently quite satisfactory, but without any signs of inflammation or general disturbance the parts simply refused to unite and separated throughout. When the anæmia had been overcome the operation was repeated in August and was at once completely successful.

Congenital syphilis is also unfavourable, and when suspected should be efficiently treated before operating (fig. 2).[1]

[1] All the drawings have been made from the actual patients and with the aid of casts, and I am much indebted to Mr. S. A. Sewell who has devoted great care and patience to ensure their accuracy.

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A big, healthy, vigorous child with a cleft limited to the soft palate might be successfully operated upon at say six months, whereas a feeble child, anæmic and with thin tissues, would probably do badly, although the operation itself might be easy.

Only too often a child suffering from cleft palate and not thriving is brought for operation on the assumption that the retarded health is consequent upon the deformity. Cleft palate is never *per se* the cause of failure of nutrition, and the surgeon must see that he is not persuaded by specious arguments to undertake an operation at the worst time.

My own practice is to operate on clefts limited to the soft palate, or little more, within the first twelve months. When the cleft is more extensive I operate on the easy cases at about two years, but I do not hesitate to defer operation on the difficult cases till as late as the sixth or seventh year. In no case is the operation undertaken until the child is in a good state of health, and indeed it is often deferred for weeks or months for that reason alone. In connexion with this aspect of the subject, it is important to recognize that many patients have done very well both from an anatomical and functional point of view when the operation has been deferred until adult life.

The General Results.

Total number of cases	155
Deaths from operation	4
Death later (ten years)	1
Patients under six years of age	22
Recently operated upon	12
Not yet traced	26
Number available for estimation of anatomical result	134
Number available for estimation of functional result	90*

* Of this number eleven have reported by letter or through their medical attendant. The remaining seventy-nine have been seen and examined by myself.

I regret that there are so many cases which I have not as yet been able to trace.[1] Those who have had experience in looking up the after-histories of their cases will know that it is an easy matter to find the first 50 per cent.; the next 25 per cent. are more difficult, and to reach the last 25 per cent. may be almost impossible. To trace the residue is not merely a question of writing letters to the patients, but many letters to relations and friends and often to doctors, and frequently several personal visits are entailed in the search. I know that some of the patients who are excluded from my tables speak quite well but I have not seen them sufficiently often or sufficiently recently to make it justifiable for me to include them for the present purpose. These remarks apply equally to a good many of the patients who have not yet attained the age of six years.

The Anatomical Results.

For this ascertainment 134 cases are available, a much larger number than for the estimation of the functional results, partly for reasons which are obvious from perusal of the table, and because in many of the twenty-six cases which cannot at present be found, the anatomical result is already known.

The gross results as to union or otherwise are shown in the diagram (fig. 3), but what cannot be illustrated is the length, the mobility, and the sensitiveness of the palate, and further, whether or not the palate, however good it may be, can take an effective part in the closure of the naso-pharynx. The mechanism of the closure of the naso-pharynx is a fascinating subject, the importance of which has only dawned upon me since this inquiry began.

The Functional Results.

In attempting to classify the cases I find myself in considerable difficulty, because there is no recognized standard by which they can be judged.

The following table sets out the results in the ninety cases which are available for this purpose:—

Normal	Good	Fair	Bad	Unintelligible
10	35	25	17	3

[1] Since the date of the meeting I have been able to trace five more of the cases—four are completely successful and one is a partial failure.

So that half the cases may be said to have good speech, and the other half bad.

Shortest time since operation	...	...	1 year
Longest " " " ...	...	...	24 years
Average " " " ...	...	...	6 "

By "normal" speech I mean that no one would ordinarily detect that the patient had ever suffered from cleft palate (fig. 4). By "good" I mean that the patients can quite easily make themselves understood, and can say all words intelligibly though there may be difficulty with some few combinations of words (fig. 5). By "fair" I mean that the patient has considerable difficulty in pronouncing some words, and though the patients may be readily understood by those who are accustomed to their disability, they are not ordinarily understood without close attention on the part of the listener (fig. 6). "Bad" speech means that the patients have great difficulty in saying most words, and those who are classed as "unintelligible" seem to have only one sound for everything (fig. 7). In these last cases I think it is highly probable that there is some defect depending upon the nervous mechanism and speech

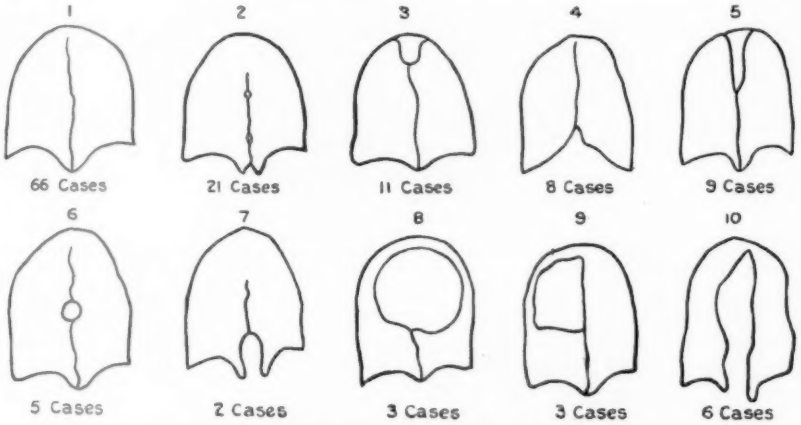

FIG. 3.—THE ANATOMICAL RESULTS IN 134 CASES.

1—Complete restoration of the palate.
2—The palate is united, but one or more pin-head holes persist or the uvula is slightly bifid.
3—There is an unclosed area just behind the alveolus. The remainder of the palate is completely restored.
4—The whole palate is shortened. There is no uvula and the soft palate is in the form of a "V."
5—The greater part of the hard palate is not united, but there is very little separation. The soft palate is perfect.
6—A hole of pencil-size persists about the junction of the hard and soft palate.
7—Part of the soft palate remains ununited.
8—The hard palate has disappeared as the result of sloughing, but the soft palate is united.
9—One-half of the hard palate has sloughed, but the soft is soundly united.
10—The palate has failed to unite in its whole length and there has been some sloughing of the edges.

co-ordination, and not merely or mainly the result of the deformity. It is well known among those who see considerable numbers of cleft-palate patients that the condition is often associated with a low standard of mental development, and, of course, this has a considerable bearing on the functional results. Some patients speak much better at times than at others, and they often have the most difficulty when they are most anxious to speak well. Nearly all have to speak rather slowly, and only those who are classified as "normal" can speak without a hitch, in a loud voice. To a certain extent the speech result depends upon the standard to which the particular patient has been accustomed, and some, for instance, speak well in the dialect

FIG. 4.—"Turnbull," age 8. Cleft of soft and posterior third of hard. Operation as out-patient at 1 year and 9 months. Now perfect palate and normal speech.

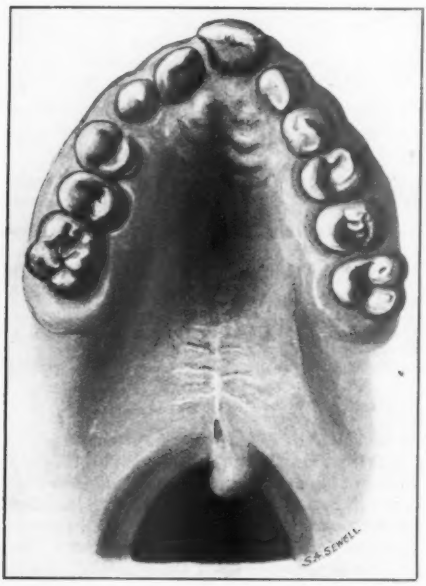

FIG. 5A.—Shows the height of the anterior part of palate in this case.

FIG. 5.—"Bainbridge," age 13. Complete cleft. Operation at 4 years. Drawing made nine years later. Speech good.

FIG. 6.—"Chambers," age 9. Complete cleft. Operation at 5 years. Hole of pencil-size at junction of hard and soft healed spontaneously. Now soft palate perfect. Speech fair.

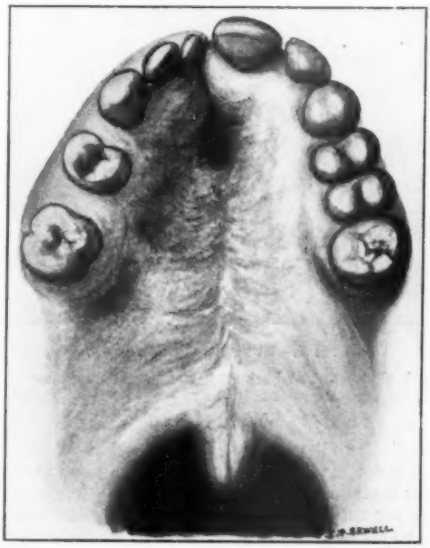

FIG. 7.—"Parker," age 8. Complete cleft closed at 3 years. Now palate mobile and sensitive, but speech unintelligible.

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peculiar to certain parts of Northumberland and Durham; whereas in London they might be looked upon as subjects of very defective speech. It is very encouraging to be able to state that in the majority of the patients whom I have been observing over a period of several years, sometimes as long as fifteen or twenty, the speech has steadily improved, though, of course, the improvement is most marked in the earlier years.

Figs. 8 and 9 show these results in more detail.

FIG. 8.—FUNCTIONAL RESULTS.

Forty-eight clefts from middle of hard palate or of soft palate only. Normal speech, 9 cases; good speech, 19 cases; fair speech, 7 cases; bad speech, 11 cases; unintelligible, 2 cases.

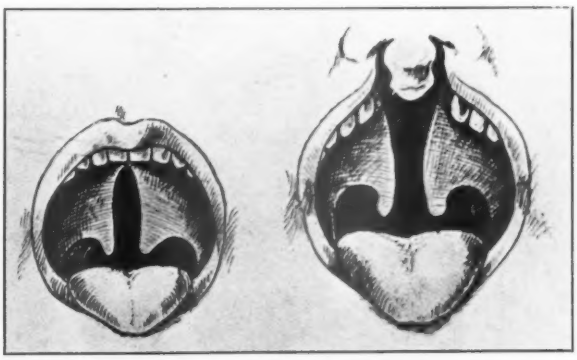

FIG. 9.—FUNCTIONAL RESULTS.

Forty-two clefts to back of alveolus and complete clefts. Normal speech, 1 case; good speech, 16 cases; fair speech, 18 cases; bad, 6 cases; unintelligible, 1 case.

Recognizing that practically all patients after operation for cleft palate speak with a marked alteration in the voice, I determined to try the plan, so frequently advocated, of restoring the palate by the method of lateral approximation (Fergusson) before the patient had ever learnt to speak. To my surprise I found that the operation was comparatively easy and encouragingly successful so far as the anatomical result was concerned, but I soon learned that one could not assume that perfect speech would follow even the most successful restoration of the palate even at a very early age. Among patients operated upon under one year and up

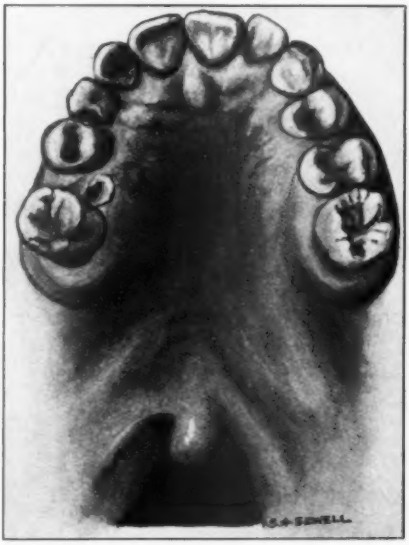

FIG. 10.—"Candlish," age 9. Cleft from middle of hard palate to uvula repaired at 2½ years. Palate now mobile and sensitive, but speech still of cleft palate type.

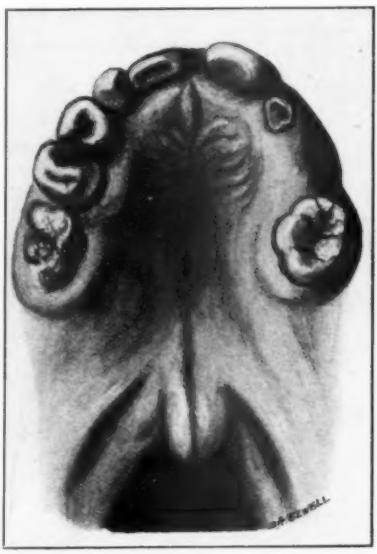

FIG. 11.—"Stephenson," age 8. Cleft of soft palate repaired at 2 years. Now pin-head holes persist, otherwise normal. In spite of careful training, speech remains of cleft palate type.

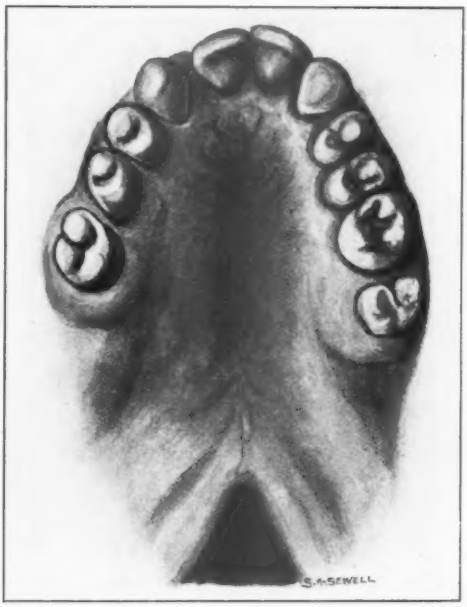

FIG. 12.—"Wilson," age 18. Cleft from middle of hard palate. Two operations at 12 years. Drawing made six years later. Speech good.

FIG. 13.—"Atkinson," age 21. Complete cleft unsuccessfully operated upon in infancy. Repair at age of 13. Palate now sensitive and mobile. Speech good.

to two years of age, there are only three who can as yet be looked upon as normal, and there are some operated upon early with perfect anatomical success who speak quite badly (figs. 10 and 11). On the other hand many of the patients on whom I have operated later than twelve years of age have succeeded in speaking very well (figs. 12 and 13). I think the good results in adults are largely due to the fact that these patients are sensitive about their condition recognizing the limitations which their defective speech imposes, and have thus tried very hard to overcome it. Not infrequently, the question of matrimony has been looming in the background, and, under these circumstances, they have been prepared to devote any amount of time to their own re-education, and they have often done so with very marked success (fig. 14).

What is the reason for defective speech in cases in which the operation on the palate has been anatomically successful? I believe that it is largely a question of the architecture, both bony and muscular, of the naso-pharynx. The closure of the latter does not merely depend on the apposition of the soft palate to the post-pharyngeal wall, but on the construction of the

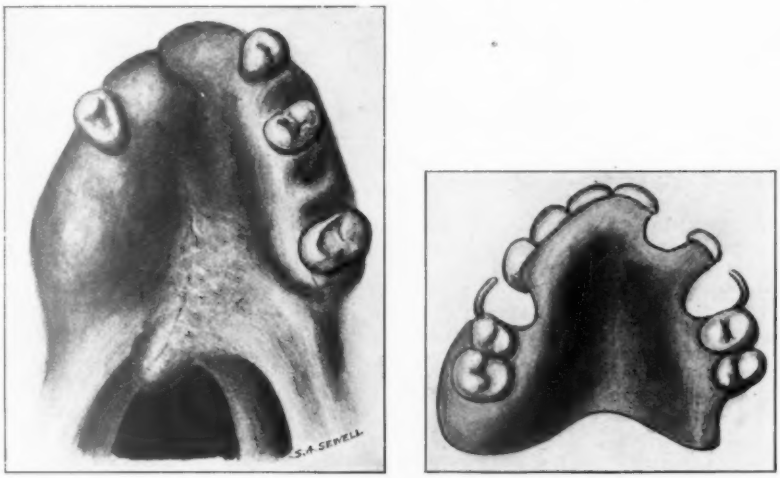

FIG. 14.—"Pitt," age 32. Complete cleft repaired at 29 years. Now good, movable, soft palate. Denture only worn to supply teeth. Speech good.

whole of the muscular apparatus surrounding the naso-pharynx. In many cases a circular sphincter can be demonstrated at the upper part of the pharynx just behind the soft palate. This sphincter is often observed in action when the patient strains during cleft-palate operations, and I am able to show you a picture of its situation from a dissecting room subject (fig. 15). Observations on cases operated upon for cleft palate show that those who talk badly have invariably a naso-pharynx which is wide and deep, and is sometimes startlingly so. This is borne out by an examination of the patients who speak well even with a bad repair of the palate or without repair, for in them the naso-pharynx is narrow and is so surrounded by thick, active muscle that it readily closes up. This, however, is an aspect of the subject which will be dealt with by my colleague, Mr. W. E. M. Wardill, who has suggested that it may be possible by local interference with the naso-pharynx so to improve the efficiency of the sphincters as to make it possible for many of these patients to speak ultimately very well, or even normally. This is a line of investigation on which Mr. Wardill and myself hope to concentrate, unless indeed those who have practised the operation introduced by Mr. Gillies will be able to prove to us at this meeting that the speech can be rendered perfect by the plan which he has advocated.

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The relationship of the anatomical result to the functional result in a small series is of great interest.

In forty-four cases in which the palate was completely restored speech was :—

Normal	... ...	in 7 cases
Good	... ...	,, 20 ,,
Fair	... ...	,, 14 ,,
Bad	... ...	,, 3 ,,

(These are the cases available for after-history out of the sixty-six in Group I Anatomical Results [fig. 3, p. 135].)

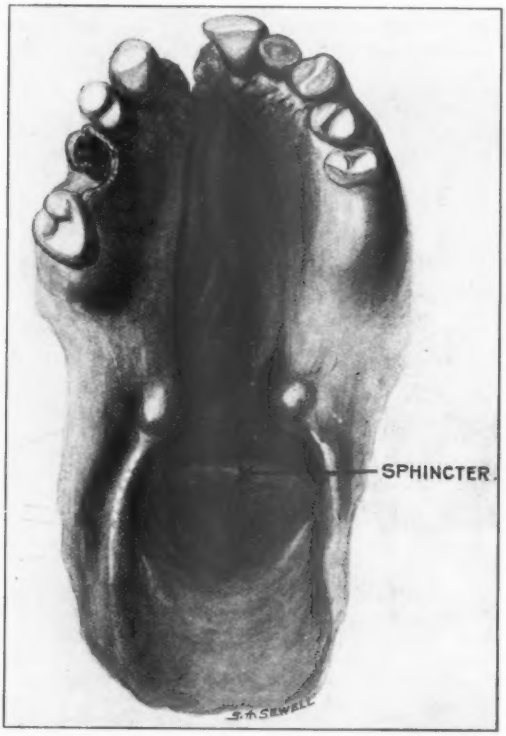


FIG. 15.—Complete cleft palate from an adult subject in the dissecting room. The pharyngeal sphincter is well shown. (Museum, College of Medicine, Newcastle-upon-Tyne. Presented by Professor Howden.)

The contemplation of these results shows the necessity of a closer study of the degree of restoration after operation, and is confirmatory of what has just been said. Evidently it is not sufficient merely to have a complete roof and a movable velum, but the whole palate must be of normal length, must be mobile and sensitive and *must suffice either by itself or together with the pharyngeal muscles to close completely the route between the mouth and the nose.* If these conditions were present in all the cases classified as anatomical successes, the speech result should be uniformly good.

Comparative Merits of the Various Operations.

I am unable to speak of the respective value of the various operations that have from time to time been advised for the surgical treatment of cleft palate, as I have always adopted the principle of the Fergusson method. We ought to aim, not only at the construction of a new roof to the mouth, but at a complete restoration of the neuro-muscular apparatus of the palate. So far as the method of Gillies and Kelsey Fry is concerned, I agree that the most important consideration is to secure a good soft palate, and that everything should be sacrificed to bring this about. For instance, if the anterior part of the cleft is wide and its repair is likely to jeopardize the soft palate—perhaps by sloughing—then it should be left to be closed by a denture (fig. 16).

The Operation in Stages.

At one time this plan appealed to me and I thought that there were considerable advantages, and especially that there would be less risk of losing tissue by sloughing. With extended experience I am now definitely in favour of attempting the whole operation in one stage, but always provided that the soft palate takes precedence, that is to say, that nothing which is done to the hard palate will endanger the closure of the soft palate. Bearing this principle in mind, it is sometimes necessary to leave a small gap immediately behind the alveolus in dealing with complete clefts, and very rarely it may be necessary to leave the whole of the anterior part of the cleft unsutured (fig. 17). Even if this is so, it is really of little importance, because these unhealed areas in the front can be so readily covered by a dental plate, and in the presence of a well restored soft palate the functional result will probably not be interfered with. I must say that I have lately found that by paying more attention to the separation round about the extreme anterior part of the cleft, it is nearly always possible to get it together without undue tension and without detriment to the soft palate.

In re-operating in cases in which the hard palate only has been previously repaired, I have usually found that the anterior part of the soft palate has been so much drawn upwards and forwards by scar tissue that in order to separate it for the purpose of suture, it has been necessary to undo the union in the hard palate, the whole operation having in consequence to be repeated. For these reasons, therefore, I have decided that it is practically always better to attack the whole operation in one stage. If, for some special reason, a two-stage operation is considered necessary, I would certainly recommend the repair of the soft palate in the first instance, as it is undoubtedly easier to repair a defect in the hard palate than to make a soft palate successfully where the hard has been first dealt with. Of course, this remark does not apply to cases where there is merely a bifid uvula or a cleft only a little more extensive.

The Question of Lateral Incisions.

I never make the lateral incisions a primary part of the operation, but invariably set out with the idea of avoiding them if possible. But one must admit that short lateral incisions have often to be made, either to enable the edges to be brought into apposition, or to relieve what appears to be an undue amount of tension after the sutures have been tied. The smallest lateral incision that will suffice is all that is ever made, and if a lateral incision on one side will serve the purpose, a second one is always avoided. The lateral incisions are made as Sir James Berry directs. Lately I have frequently employed what I call tension scratches, which are merely superficial incisions through the mucous membrane and perhaps a few fibres of the underlying muscles without penetration of the palatal tissue itself. These tension scratches are made if required in the positions shown in the illustration (fig. 16).

I have always feared free lateral incisions, because it appears to me that if they fail in their purpose, a large area of the palatal flap is likely to be lost by sloughing and can never be replaced. If, on the other hand, only a small lateral incision is made and union fails, the tissues nevertheless remain, and the operation can be repeated with a reasonable chance of success. This is borne out by the record of my own cases in which I have often carried out such secondary operations with complete success. I have never seen any difficulty from the short lateral incisions and there has been no case in which the incisions have failed to heal. Upon my examining the palates afterwards I have never encountered a scar that has been so marked as to be likely to interfere with the mobility of the palate. I have

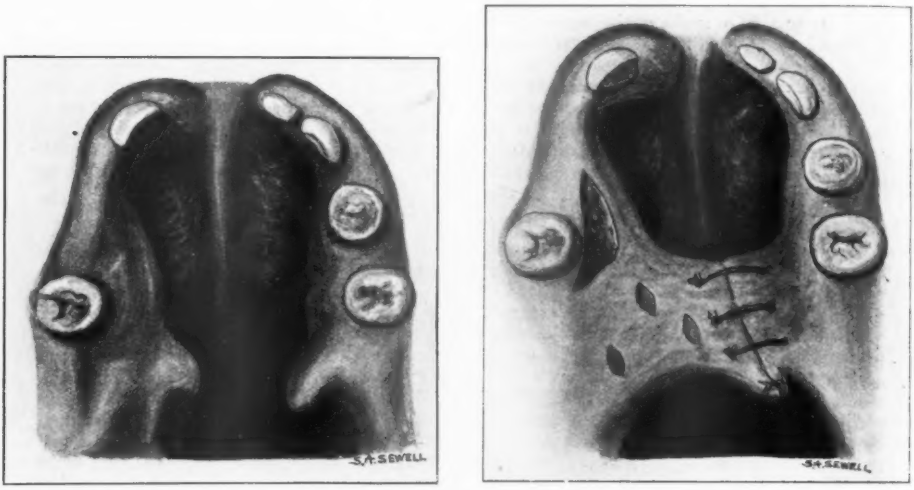


FIG. 16.—"O'Connor," age 23. Three previous operations had been failures. Attempt to repair soft palate, October, 1925. Note lateral incision and tension scratches.

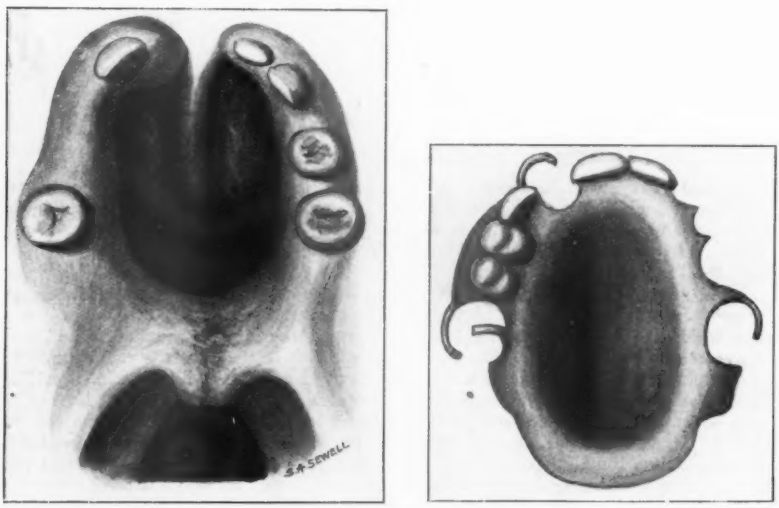


FIG. 17.—"O'Connor" (same case as fig. 16), age 25. Condition of palate, April, 1927 (i.e., 2½ years after last operation). With the aid of the obturator the patient speaks quite intelligibly.

FIG. 18.—"Gordon," aged 5. Shows the palate three years and four months after operation. The boy wears an obturator to cover the defect in front. As yet speech is bad; he has had no special training.

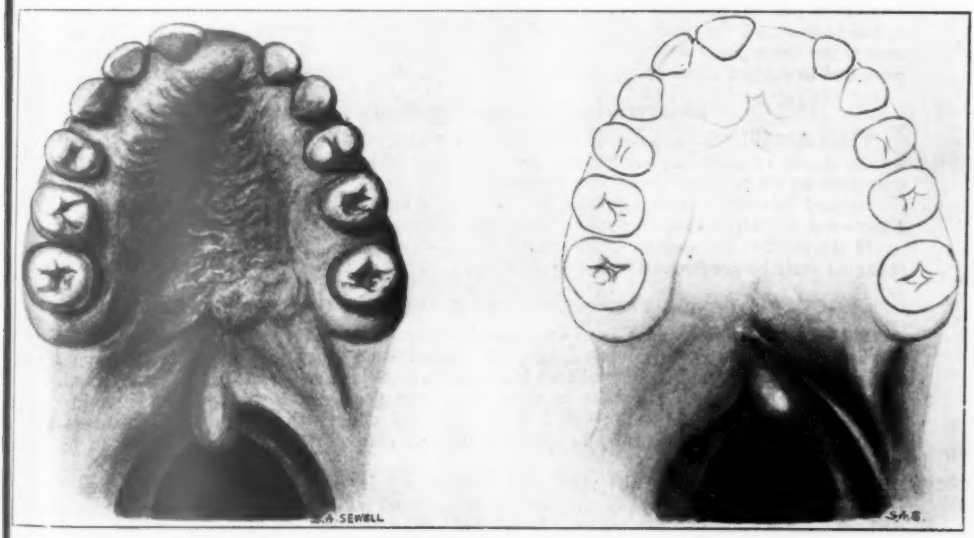


FIG. 19.—"G. Allen," age 21. Complete cleft unsuccessfully operated upon at 8 years. Repair at 18 years. Very difficult on account of dense scarring. On right side free lateral incision, much smaller one on left. The drawing shows the result three years later. Left side retracts actively on stimulation, but the right hangs flaccid. Speech is fair.

been greatly struck by the very small sign of the lateral incision which remains. Similarly, I have not been able to connect the failure to secure a good functional result with any damage that may have been done by such small lateral incisions. In a good many of the re-operations, where the primary operation has been performed by surgeons who have used extensive lateral incisions, I have encountered a large amount of scar-tissue and have experienced great difficulty in separating this from the bony palate. In one or two cases I have also noticed that on movement, the palate has been pulled more to one side than to the other, and this may have been the result of more extensive or deeper lateral incisions on the quiescent side (fig. 19). But, on the other hand, I have noticed the same unilateral pull of the palate in cases in which lateral incisions have not been employed.

A lateral puncture is a useful avenue, sometimes indispensable, for the introduction of such instruments as separators. In certain difficult cases I have had to employ more than one such puncture on either side, and at the end of an operation where they have been employed I have not infrequently enlarged the puncture so as to make it into a short lateral incision. Such punctures or short incisions for the introduction of instruments are very frequently required in secondary operations.

Sutures and the Method of their Insertion.

So far as the suturing is concerned, I do not rely on any special method. I have at various times adopted different plans, such as the continuous stitch and the figure of eight suture, and I have used supporting sutures of various kinds, but I am not convinced that any of these methods have been of real advantage, and I have therefore discarded them. If the parts can be got into easy apposition without tension, that I think is the best criterion of success, and no special method of suture will compensate for the want of it. In practice I use interrupted sutures, passed with an ordinary small needle such as that recommended by Sir Arbuthnot Lane. The suture material is chiefly silkworm gut, rather thicker for the two or three main sutures, and quite fine for the intervening stitches. I have always used fine 6/0 chromicised catgut for the upper surface of the uvula, and I have recently used it for some of the intermediate sutures and for the closure of the anterior part of the palate. It has been most efficient and has this advantage, that it does not require to be specially removed. It has been stated that catgut is absorbed too quickly in the mouth, but this is certainly not true of the chromicised variety, in fact, I think it is rather the other way. At all events, I have had to cut out a fine catgut stitch in July which was introduced into the palate in April.

Preliminary Treatment before Operation.

There should be no obvious septic focus in the mouth, and carious teeth or much infected tonsils should be removed, but this must be done three or four weeks before the contemplated operation on the palate, in order that the wounds may be healed.

Beyond attending to such obvious foci I make no special preparation of the mouth, and I have not, as yet, carried out routine bacteriological examinations.

If the child is old enough to be taught to wash out the mouth or allow it to be sprayed, it should early be accustomed to the procedure.

Dental Treatment as an Adjunct to Operation.

This is often helpful, and I have employed it a good deal, and especially since I have had the help of Dr. Elsie Peet, who has carried out the necessary procedures with much patience. The proper restoration of the dental arch is of considerable help so far as speech is concerned, and even if it is impossible to make anything like a perfect arch, at least it is most helpful to supply incisors and lateral teeth; this I have often had done with considerable advantage. Similarly, the wearing of dentures to cover an anterior defect in the palate has often to be adopted (fig. 16).

Dental treatment during the process of healing of holes is also helpful; without doubt many anterior defects or holes further back in the palate heal more quickly under plates than without their assistance. But we have found that it is of little use attempting to fit temporary plates in the mouths of small children. The real field for dentistry is in the later stages of the management of these cases, that is to say, when the treatment comes to be "finalized" —an ugly but expressive word which I think is particularly appropriate in this connexion.

SPEECH TRAINING.

As yet only a small proportion of my patients have had the advantage of special training so I have very little experience of the results. I have, however, noticed that if a patient with a well-repaired palate reaches years of discretion and has not learned to speak reasonably well, then even expert tuition does not greatly improve their efforts. Probably to get reasonably good results, and certainly to get the best results, it would be necessary for the patients to undergo speech training from the moment they attempt to speak after the operation. It would be helpful if those engaged in this work of training could investigate those cases of cleft palate in which the patients have never been operated upon, but who speak quite well. A study of the varying degrees of defective speech in those in whom the anatomical repair has been satisfactory ought also to be instructive.

SECONDARY OPERATIONS.

Twenty-four of my patients had previously been subjected to operation by other surgeons, and I have had to re-operate, often more than once, in thirty cases where I was responsible

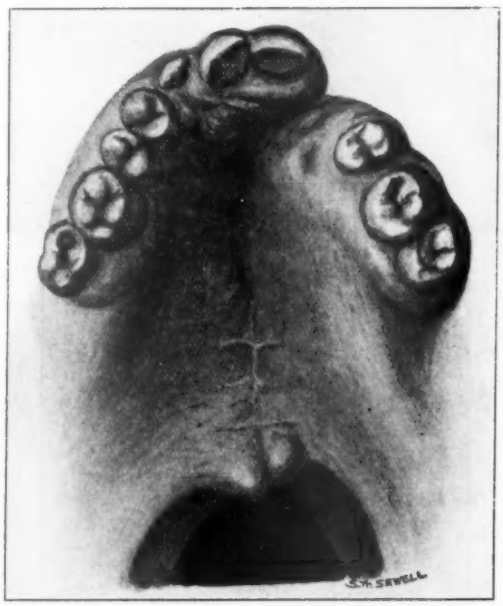
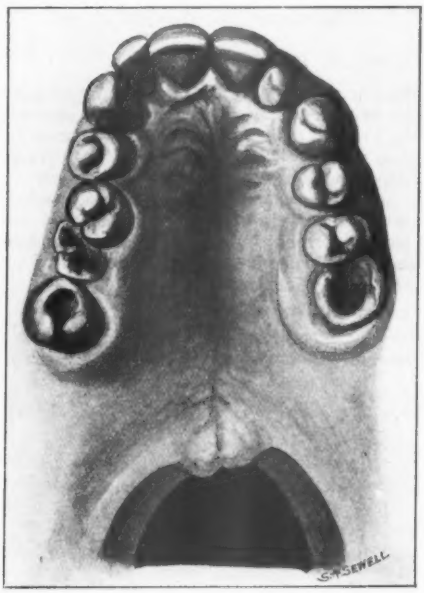


FIG. 20.—"Curry," age 15. Complete cleft. Unsuccessful operation at 3 years. Drawing two years after repair done at age 13. Soft palate movable and sensitive. Speech good.]

for the first interference. The difficulty of these operations and the prospect of success turn almost entirely on whether or not there has been actual loss of tissue by sloughing.

Retracted, distorted and densely adherent palatal tissues may be coaxed into apposition in a surprising way, but I know of no method by which tissue actually lost can be successfully replaced, although I am looking forward to hearing this evening something of Mr. Gillies' successes in this respect. Short of this loss of tissue, the more complete the previous failure has been, the easier will be the secondary operation. In fact, when the hard palate has been successfully united the soft palate is usually so drawn forwards and fixed that the whole has to be undone in the hope of making at least a soft palate sufficiently long and mobile. In these re-operations I have always concentrated on the restoration of the soft palate, though I have often succeeded in making a satisfactory closure of the whole (figs. 20 and 21).



[FIG. 21.—"Sanders," age 17. Cleft from alveolus. Drawing made eight years after last of four operations. Soft palate mobile and sensitive. Speech fair.

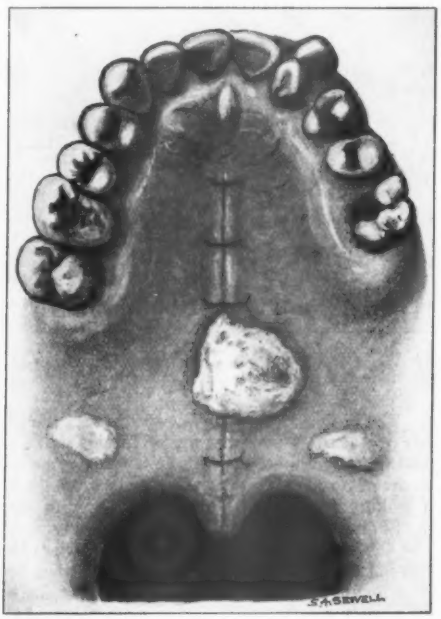


FIG. 22.—"Gaymer," age 13. To show slough about the central point of the suture line four days after operation. After separation a large hole remained.

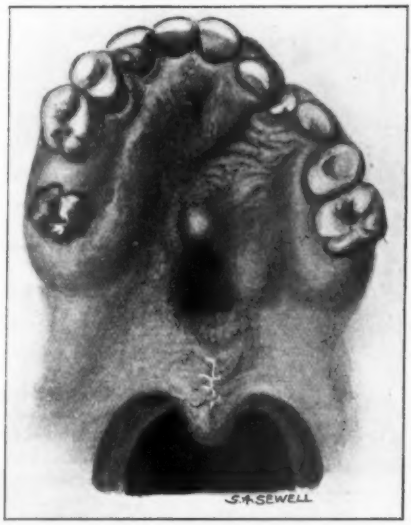


FIG. 23.—"Wilson," aged 6. Complete cleft. Lateral incision left side. Shows hole at fourteenth day. Rapid healing.

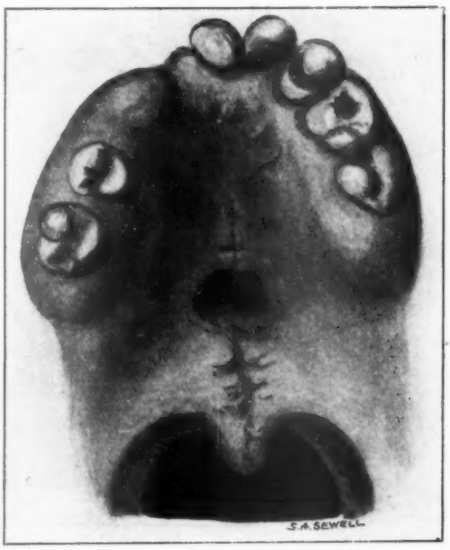


FIG. 24.—"Faulkner," age 12 years and 11 months. Complete cleft. One small lateral incision. Shows hole ten days after operation. Rapid healing.

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FIG. 25.—"Lodge," age 7. Cleft from middle of hard palate. Operation at 3 years, central hole—of the actual size shown in the outline—healed in three months. Drawing of healed palate made four years after operation. Palate mobile and sensitive. Speech good.

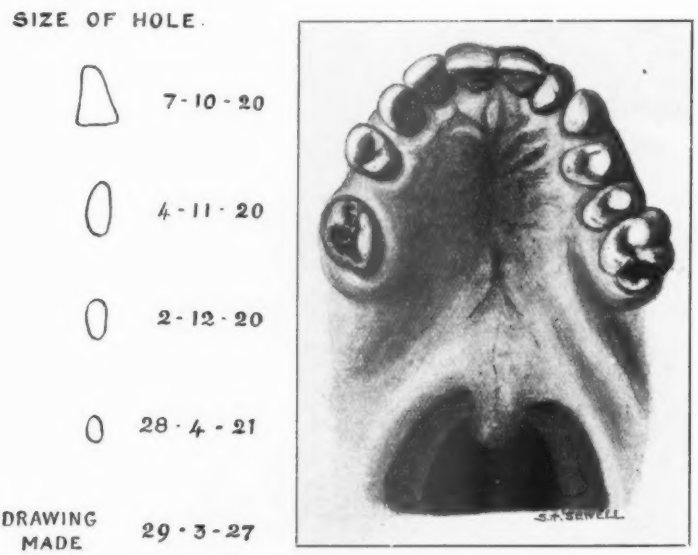

FIG. 26.—"Cook," age 16. Cleft from middle of hard palate, operation at 10 years, August 21, 1920, followed by hole of the size shown in the diagram. Now mobile and sensitive palate. Speech fair, and steadily improving.

Other types of secondary operations may be required for the closure of central holes or of an anterior gap, or for repair of the uvula. The latter seldom presents any difficulty, and, in my view, is always worth careful attention, as it adds to the length and probably the functional usefulness of the soft palate. The closure of anterior defects I have found to be very difficult and often attended with only partial success. This is not very important, because such defects can always be covered by a plate or obturator, and very often this is necessary in any case to carry front teeth which are so often missing or defective when the cleft has been a complete one.

Holes in the middle of the palate belong to quite a different category. I have been distressed at the frequency with which these occur in my operations (figs. 22, 23 and 24), and am hoping to learn how they may be avoided. But I am frequently comforted by the ease with which such holes spontaneously heal without interference with the length or mobility of the palate (figs. 25 and 26). If such holes do not show signs of rapid healing, then their progress must be carefully watched. If they heal from side to side, though it be slowly, no harm will follow ; but if healing is from front to back, they are harmful and should be dealt with, because the soft palate is dragged forward, shortened and rendered more rigid. An aperture which is harmlessly but slowly closing may sometimes be hastened by covering it with a temporary dental plate.

When operative interference is necessary it will probably mean rather long lateral incisions with free separation of the tissues from the edge of the aperture. Even then the sutures which are used to approximate the edges frequently cut out, but the resulting hole is almost invariably made smaller and healing is hastened and corrected. I am now speaking of cases in which the soft palate, and, in fact, the greater part of the cleft, however extensive, is soundly united, and under these circumstances free lateral incisions need not be feared.

Most of the points suggested by the headings laid down by the Council for discussion have now been reviewed, but there are certain other matters of interest raised by my cases with which I will deal briefly.

The Relationship of the Treatment of Hare-lip to the Cleft Palate Problem.

The treatment of hare-lip has properly been excluded from this debate, but no doubt one may be permitted to say that when the operation on the lip is carried out during the first three or four months of life, it has a very beneficial effect in drawing together the anterior part of the cleft, and making its closure subsequently easier and therefore likely to be more satisfactory. At the same time it must be recognized that this early operation on the lip does most definitely narrow and distort the dental arch.

The premaxilla should not be removed, as it helps in the closure of an anterior cleft and somewhat preserves the dental arch, preventing it from falling together at the anterior end and becoming more or less V-shaped. The inefficient repair of a lip, which leaves it too flabby or with poor labial apposition, has also some slight ill-effect so far as the speech is concerned.

I would also point out how important it is to have a good external orifice for the nose, for in not a few cases I have found that a too wide anterior naris has allowed an unregulated escape of air, and has certainly contributed to the faulty speech which has sometimes been present in spite of the perfect closure of the palate (fig. 27).

Where Should the Operation be Carried Out?

In the district in which I work the difficulty of finding hospital accommodation is very great. Very young children do not settle well, in hospital, and it is usually impossible to provide accommodation for their mothers as well, and even when this can be done the environment for both is strange and often inconvenient.

These considerations led me to operate on some of these cleft-palate babies as out-patients. At first I began tentatively, and selected only the very easy clefts of the soft palate, but the patients did so well that I extended the practice to more extensive clefts and older children. Now I operate on a fair number in this way, and am sure that the patients have often done better than they would have in hospital.[1] There have been no calamities and no special

1 During the first two weeks of August I operated upon eight cases of cleft palate. Seven were treated as out-patients, and with the exception of very small holes in two cases, they all healed perfectly. One child was kept in hospital as the mother appeared to be incapable of looking after it. It was an easy case, but the child fretted and cried all the time, and without any sloughing almost the whole suture line gave way.

anxieties. Of course, cases are only undertaken when the children are healthy and the mothers appear to be careful and obedient. The patients are kept waiting in hospital an hour or two before the operation in order to ensure that the stomach is not overloaded, and they are not allowed to be taken home until it is obvious that there is no unusual amount of bleeding. The mothers are told to bring the children at the end of a week for the first inspection, or at any time before that if they have anxiety about them.

The following table gives some particulars about the twenty-three patients who have been dealt with in this way:—

CASES OPERATED UPON AS OUT-PATIENTS.

Twenty-three patients.

Youngest	8 months
Oldest	27 years
Average age of children	Just under 3 years

Two cases had to be re-sutured and in both the result was ultimately successful. The final results were all satisfactory.

FIG. 27.—Shows inefficient repair of nostril after operation for hare lip, providing uncontrolled leakage of air, thus contributing to faulty speech after successful operation on palate.

But when the child is to be operated upon as an in-patient in a hospital or home, it is best that it should be acclimatized. I envy those surgeons whose accommodation makes it possible for them to keep their cases waiting in hospital until all risk of general infections has passed, and until they are assured that the child's nutrition is good and that it is thoroughly accustomed to its environment.

Operations on Patients More Than 12 Years Old.

Twelve years is the age at which in my hospital children have to leave their wards and go among the adults, so that it forms a convenient time at which to make the distinction between little ones and those grown up.

Certain considerations must be borne in mind when operating upon adults. These patients not only become accustomed to their deformity, but they have usually acquired certain tricks of articulation which enable them to speak so as to make themselves readily understood. The more successful the operation on the palate, the more their previous speech mechanism is upset, and in consequence they often feel extremely awkward for a very long time afterwards. None of my patients have regretted having undergone the operation. But interference must be recommended with proper restraint and it would be foolish to make it general.

Some of these patients have informed me that though their friends tell them that their speech is much improved, they do not themselves appreciate the fact; undoubtedly it takes a considerable time for them to acquire facility under the altered conditions. They are, however, much more comfortable and have often said that a previous tendency to catch cold has disappeared.

In the one adult patient in whom the operation was a complete failure, so far as union was concerned, there was, strangely enough, a considerable improvement in the facility of speech. This I attributed to the fact that the soft palate was previously drawn forwards by dense scar, and in this way shortened and anchored. As the result of the operation, separation was so thorough that the halves were each rendered much more movable.

The following table gives particulars of all the patients over 12 years upon whom I have operated:—

Twenty-three cases: seven males, sixteen females.

Youngest	13 years
Oldest	40 ,,
Average age	18 years 9 months

In eleven there had been previous operations.
Only one case failed to unite.

The functional results have been very encouraging and most of the patients speak much better than before (fig. 28).

The After-treatment.

I do not think that much can be done in the days immediately following the operation that is likely to influence the result.

If the patient can wash out the mouth or will allow it to be sprayed, well and good. If not, I make it a rule that the child must swallow a little boiled water or weak peroxide of hydrogen solution after each feed in the hope that in this way the mouth may be irrigated into the patient's own stomach. Children do not bear starvation well, and I think it is important that they should have an abundance of liquid nourishment as soon as ever they care to take it. After the first week some soft pudding, such as custard or curds and whey, or bread crumbs and milk, or crumbs and gravy, may be added. At the end of three weeks the type of food will not affect the result. Nothing must be done that will make the child struggle or cry, and if mouth washing or spraying is resented, it should not be carried out. For the same reason the purgative chosen should be of an agreeable nature. Liquorice powder mixed with milk, or syrup of figs, or half a grain of calomel are all suitable.

The continued after-care of cleft-palate cases is most important, and until everything has been done that may possibly contribute to a completely successful result, the patient should be seen from time to time.

In many cases it is only after months or years that the results can be looked upon as final.

The Actual Operation.

In the conduct of this I have nothing to add to the description so admirably presented by Berry and Legg in their book on the subject.[1]

The position of the patient, the method of anæsthesia and the instruments used are matters which each surgeon will ultimately decide for himself, and until an operator has had

[1] "Hare-lip and Cleft Palate, with Special Reference to the Operative Treatment and its Results," London, 1912.

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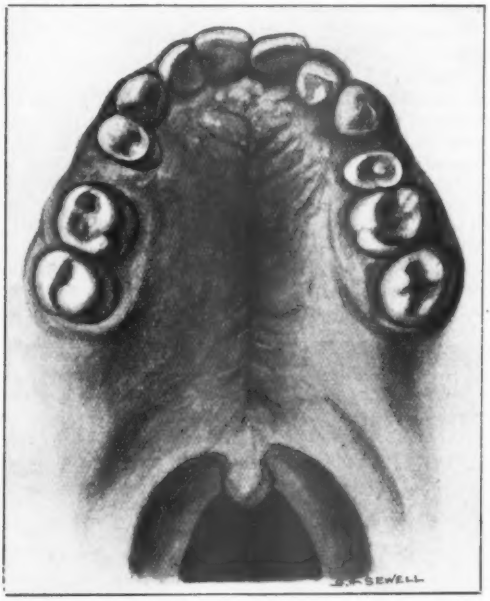

FIG. 28.—"Millar," age 25. Cleft from middle of hard palate. Unsuccessful operation at 5 years. Repair at 18. Now mobile and sensitive palate. Speech good.

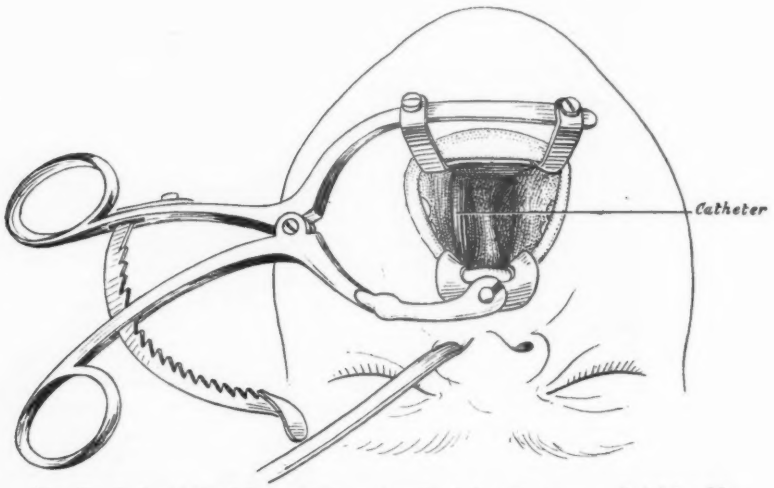

FIG. 29.—Patient in head-down position. Anæsthesia by ether-oxygen administered by catheter through nose. Von Eiselberg gag in position.

enough experience to suggest modifications, he cannot do better than follow the directions so clearly given in the work mentioned.

Personally, I much prefer the von Eiselberg gag, and I like ether given through a nasal catheter (fig. 29) from the Shipway apparatus, in which the vapour is propelled by oxygen instead of by the hand bulb. Anæsthesia by rectal ether sounds almost ideal, but in my experience it has this disadvantage, that either there is anxiety because of the depth of the anæsthesia or the patient is not deeply enough under, and still requires some anæsthetic to be given by the mouth.

The beginner must have the most scrupulous regard for hæmorrhage and must learn that a dangerous loss may easily occur. It is not sufficient to mop up blood, its escape must be *arrested* by steady sponge pressure, and the time necessary for this purpose must never be grudged. Although I can only speak from a very limited experience of its use, I must say I fear the suction apparatus, because the more efficient it is, the more readily it sucks up blood, in that way disguising what may really be a serious loss.

Conclusion.

An interest in surgical history made me anxious to establish the claims made for our own Sir William Fergusson as the first to devise and practise the operation which is so frequently associated with the name of his German contemporary, Langenbeck. In spite of kind help from Mr. Victor Parr, of the Library of the College of Surgeons, and from others, I have not been able to settle this question of priority. I feel that it is perfectly fair to speak of the method of lateral approximation as now carried out, as the "Berry operation," for it is certainly Sir James who has put it on its present-day footing. I can only date any success of my own to the time when I had the opportunity of studying the most admirable descriptions of the operation which he has published, and I have to acknowledge gratefully the extreme importance of visits which I paid to him for the purpose of seeing cleft-palate surgery in his own hands.

Mr. O. L. Addison.

One of the questions set down for discussion was on the relative merits of the flap and Langenbeck operations.

I have had a very considerable experience of both methods and do not hesitate to say that the Langenbeck gives the better results. The flap operation if done in infancy has a heavy mortality, and a large proportion of failures and incomplete closures, large holes frequently being left that are difficult or impossible to close later. The late results as regards mal-eruption of teeth, severe contraction of the alveolar arch, and dragging forward of the soft palate, are very bad.

The Langenbeck operation has a very small mortality and a greater proportion of successful closures, and causes only very slight contracture of the alveolus. It also produces a longer and more mobile soft palate and is therefore more likely to give good speech. The age at which the operation is performed appears to have little influence on speech and a perfect anatomical result does not insure good speech. Cases are frequent in which gross defects, even complete clefts, are present with almost perfect speech, while it is common to find hopelessly bad, even unintelligible, speech with a good anatomical result. It seems certain that good speech is almost entirely a matter of constant and careful training together with intelligence and determination on the part of the child. In the preparation of the child for operation the general health is by far the most important factor.

Rhinorrhœa or otorrhœa is an absolute bar to operation. Local conditions that must be dealt with are gingivitis and carious teeth. Except in the case of small infants with partial clefts the tonsils and adenoids should always be removed, they are almost constantly found to be enlarged with crypts full of caseous débris and are the main reservoirs of sepsis in the mouth.

No good results have been obtained from the use of preliminary injections of polyvalent antistreptococcal serum, but every case is Dick tested and, if positive, given an injection of antiscarlatinal serum.

The success of the operation depends on the avoidance of tension, and sepsis; the latter can only be partially provided against, but the former can be absolutely prevented by the use of long lateral incisions. These should begin on the lower jaw, and should be carried up the

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ascending ramus to the upper jaw behind the centre of the last molar, then forwards as far as necessary, immediately against the teeth. This incision, if properly made, is external to the palatine artery and to the tensor and levator palati muscles and does not interfere with the movement of the palate. The late results are excellent, in some cases better than when a short incision is made, probably because with the latter a greater degree of separation of the soft from the hard palate is necessary and therefore more injury is done to the muscles.

Tension sutures are bad in principle; because they only act for the first few days when their presence is unnecessary; after that time they have cut so far into the palate as to be of no value and merely act as foci of necrosis and infection. Similar objections apply to the use of plates which, if left in position for longer than five or six days, are apt to cause necrosis. A palate seldom breaks down till after the end of the first week.

With this method of operating, in fifty-nine out of seventy-seven cases of all types, healing took place after one operation only. Of the five partial failures most were only a few stitches that had given way, and of the eight with holes in only two or three was it worth while to consider closing them. Three cases failed completely, one after scarlet fever. In two cases the patients died, one from hæmorrhage the night after operation, and the other from convulsions.

M. Victor Veau (Surgeon to the Foundling Hospital, Paris).

Muscular Suture.

I thank Mr. Warren Low, the President, for having so kindly invited me to speak at this meeting, and I wish to pay a special tribute to Sir Arbuthnot Lane from whose works, in operating according to his method, I have learnt most, in this difficult surgery of the cleft palate.

At the outset of my professional career I operated according to Langenbeck's method, introduced into France by Trélat. In 1922[1] I gave the results in eighty-nine cases of children operated upon by this method:—

74 per cent. total closure with one or several operations.
13 per cent. incomplete definite closure.
3 per cent. total failure.

The anatomical result is of no value, the phonation only is the criterion of the operation, because we do not operate to close an aperture which nobody sees! Out of forty children operated upon, seen some years after the operation:—

25 per cent. spoke well soon after the operation with little or no education, but not one spoke normally;
15 per cent. had been improved by a phonetic education continued over a long time;
60 per cent. had received no benefit whatever as regards speech.

I maintain that the bad functional result is due to the method of operation which often results in producing short and immobile palates. The anatomical insufficiency of Langenbeck's palates is chiefly due to the sclerosis caused by the cicatrization of the large bleeding surface on the nasal aspect of the palate in a part which is not seen. It is the fatal consequence of the fact that Langenbeck undermined widely *in order to lower the flaps;* this is the cause of all the trouble.[2]

Impressed by the bad result of the classical method I followed that of Arbuthnot Lane in six cases. I obtained a very good result, an account of which I presented in 1921 at the Surgical Society in Paris.[3] I learnt very much in carrying out this method, because I saw that in the unilateral form which is the most frequent, the mucous membrane of the vomer makes a very excellent flap which Langenbeck did not know how to use. The method which I propose is derived from that of Arbuthnot Lane.

1 *Bull. et Mém Soc. de Chir. de Paris*, 8 Mars, 1922, 357.

2 I realize that in France our palates are probably more sclerosed than those in England, because at the stage when the mucous membrane is cut with English scissors we bruise it between the fingers and a rugine. This practice was handed on to me by Erhmann and Jalaguier.

3 *Bull. et Mém. Soc. de Chir. de Paris*, 1921, 299.

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(A) Principles of the Method.

(1) *Total suture of the nasal mucous membrane* on the upper surface of the reconstructed region. Not only do I abstain from making transverse incision of the nasal mucous membrane along the posterior edge of the palatine bone, but I carefully close the median opening in order to prevent the nasal mucus from passing through this opening.

(2) *Muscular Suture.*—I do not cut any muscles. The cutting of muscles is an irrational practice in the case of an organ which exercises its functions only by ready mobility.

For the closure of the palate I depend on the application of a strong copper wire embracing all the muscles which, as previously mentioned, have not been cut. I do not fear the tension of this suture: my wire is stronger than the tension (figs. 1 and 2).

(3) When the opening extends to the bony palate I completely liberate one or two buccal flaps. But I do not free them in order to sew them like a bridge according to Langenbeck's method, by which they are freed for the purpose of being lowered. My practice is to liberate the flaps in order to move them as a drawer is moved. I suture them at the apex of the palatal dome; in this way I avoid the dead space of Langenbeck.

In 1922 I published in detail in the *Journal de Chirurgie*[1] the anatomical facts upon which this method is founded.

(B) Technique.

I will now briefly relate the different steps of the operation, based on an experience of 320 cases.

(*I*) *Cleft of the Soft Palate.*

First Step (fig. 3).—The beginning of the muscular suture.

The needle is introduced externally to the anterior pillar, internally to the mandible. It is at once pushed up to the superior surface of the velum where it continues its course between the muscles and upper mucous membrane. The needle comes out at the level of the free edge. It is threaded with a copper wire three-tenths of a millimetre thick.

(Fig. 4.)—The same process is carried out on the other side, but on this side the needle is threaded with a double silk thread which, after the nasal suture is completed, will be utilized for drawing the copper thread round the muscles on this side (fig. 7). It is preferable at first not to pass the copper thread on *both* sides because it would be too much in the way of the suture of the nasal mucous membrane.

This step is the most important. It is essential that the thread should be passed exactly beneath the upper mucous membrane of the velum. No muscular fibre should be left between the needle and the mucous membrane. The value of the muscular suture depends entirely on the exact passage of this thread.

Second Step (fig. 5).—The freshening of the edges of the cleft is carried out as in every method.

Third Step (fig. 6).—Nasal suture. I am accustomed to make this suture with a small Reverdin's needle. The sutures are made in such a way that the knots are on the upper surface. I take no particular precautions in placing these stitches.

(Fig. 7.)—The completion of the suture of the nasal mucous membrane. The suture of the nasal surface of the uvula is difficult because, here, the membrane has no consistency.

The muscular suture is continued. The inner end of the copper thread is introduced into the loop of the silk thread which has already been passed on the left side. In pulling the silk thread the copper wire is conducted to the other side, and so on both sides the copper wire now lies immediately beneath the nasal mucous membrane. You can see it in the middle of the cleft.

(Fig. 8.)—The completion of the muscular suture. The needle is passed *under the buccal mucous membrane* from within out to the primary entry point of the copper wire. It is then withdrawn, bringing the copper wire into the median wound. In this way the copper thread forms a complete circle—under the nasal mucous membrane above and under the buccal mucous membrane below. The circle of the thread embraces the whole of the muscles of the velum.

It is very easy to pass the needle under the buccal mucous membrane because it is seen. The difficulty is to pass it under the nasal mucous membrane because it is not seen. The only help to be derived is by placing the forefinger on the upper surface of the velum.

[1] *Journ. de Chirurgie*, July, 1922, 1.

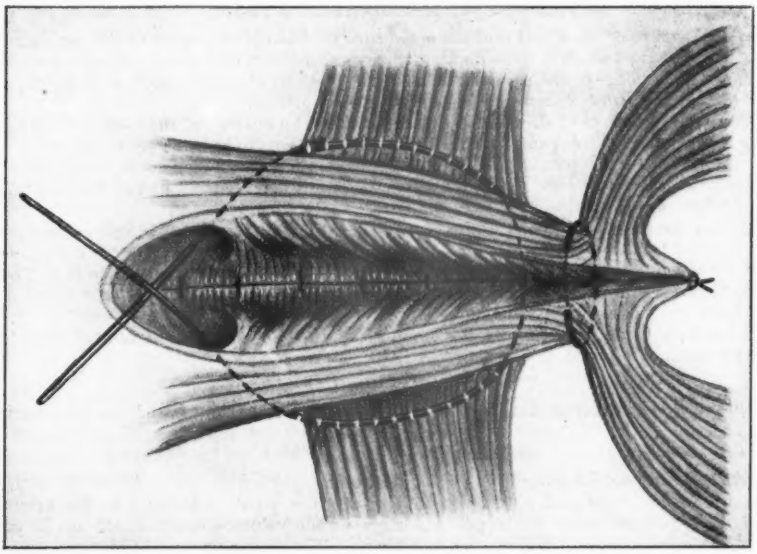

FIG. 2.—Muscular suture.

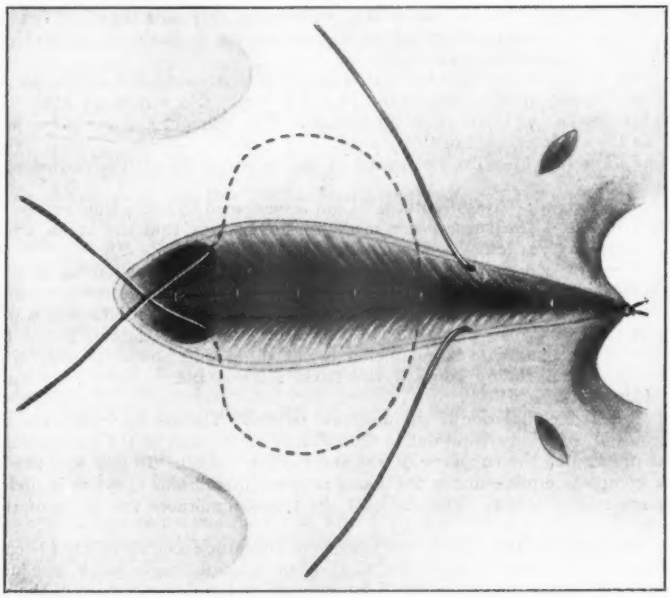

FIG. 1.—Muscular suture.

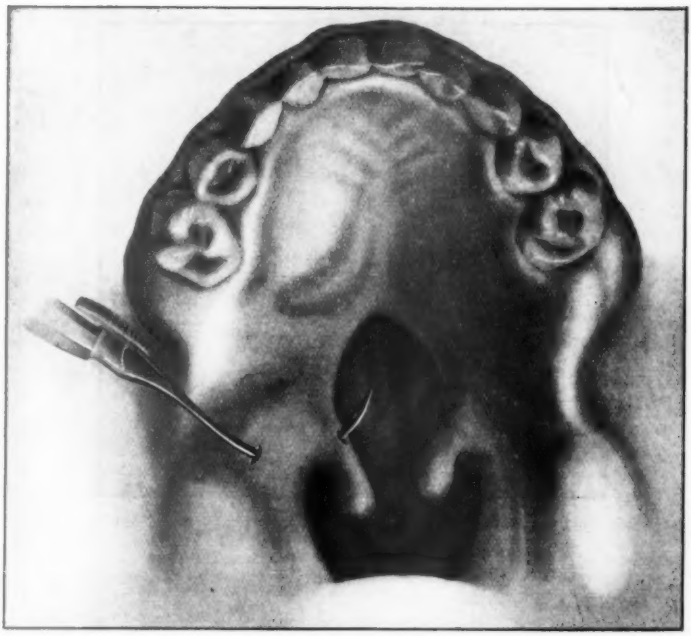

FIG. 3.—Muscular suture. The needle runs just beneath the nasal mucous membrane, its concavity includes all muscles.

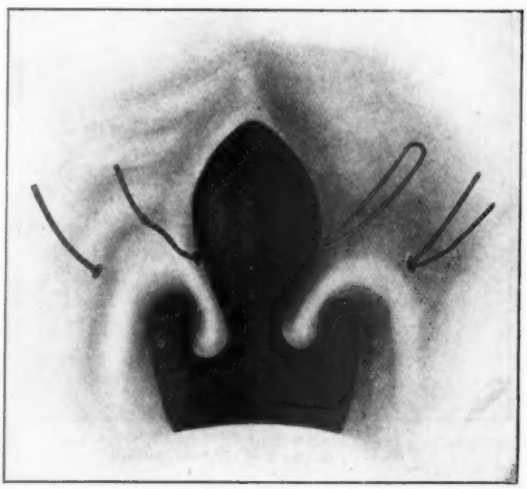

FIG. 4.—Both threads are passed, on the right copper, single; on the left silk, double.

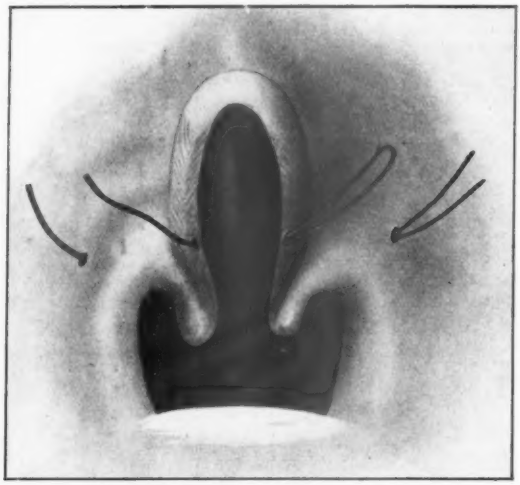

FIG. 5.—The freshening of the edge.

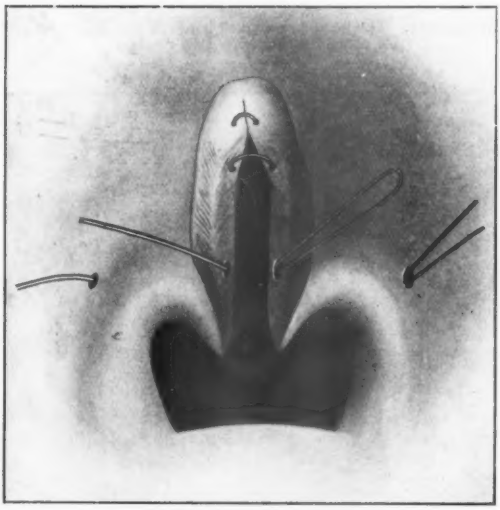

FIG. 6.—Beginning of the nasal suture.

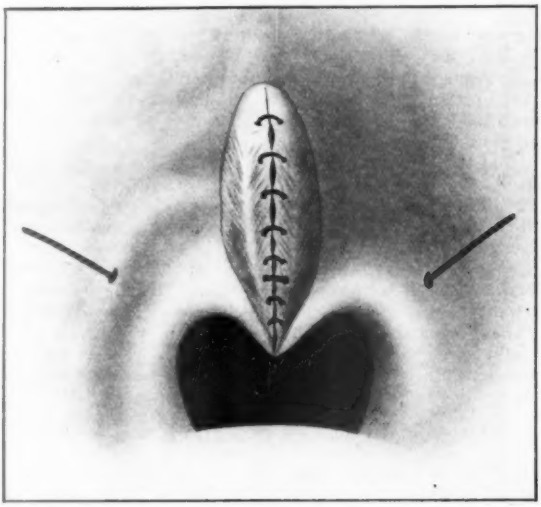

FIG. 7.—End of the nasal suture. The copper thread is passed on both sides under the nasal mucous membrane.

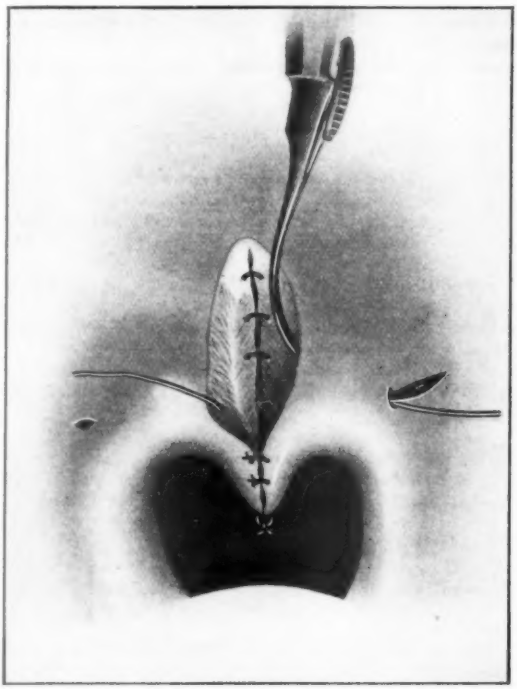

FIG. 8.—The copper thread is passed under the buccal mucous membrane.

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(Fig. 9.)—The copper thread is now tightened. I pull the thread very tightly. There is more to be feared from not pulling tightly enough than from the contrary action. There is no risk of cutting through the muscles by pulling too tightly, except in the case when the muscles have not been properly included in the metallic loop. Experience has shown me that when this happens it is only on the left side, because on this side the needle is passed through with much more difficulty.

You will notice that the loop of the copper thread does not take any segment of the buccal mucous membrane.

The toleration of the wire depends on the observance of this point of technique. It is left in for eight to fifteen days. It is difficult to remove. On five occasions the loop has been left in permanently without detriment.

Fourth Step (fig. 10).—Buccal suture. The buccal mucous membrane is sutured as in the classical method. I attach no importance to this. In Langenbeck's opinion this suture was everything. In my view it is almost nothing, because I have over it a mucous membrane roof which is very complete, and above all a muscular suture which has much more resistance and which alone ought to be sufficient to ensure the union. As a matter of fact, I have never experienced any accident in dealing with clefts of the soft palate only.

(*II*) *The Cleft of the Soft and Hard Palate.*

If the cleft extends beyond the posterior edge of the palatine plate, success is less certain, because at the level of the bone no muscles can be sutured (fig. 11).

In front it is impossible to suture the buccal mucous membrane. Two buccal flaps must be liberated, but I do not, like Langenbeck, free them in order to lower them, but to raise them. They are fixed to the roof by two stitches which include the nasal mucous membrane (fig. 12). Langenbeck had a large irregular bleeding surface on the upper invisible aspect of his flaps. I have a smooth visible surface in front of these flaps, a surface which is swept by the movements of the tongue.

It is necessary carefully to preserve the palatine artery: this is not at all difficult, because you see the artery in raising your flaps.

(*III*) *Unilateral Cleft with Hare Lip.*

It is here that I carry out the same technique as Arbuthnot Lane, that is. I turn up a flap taken from the vomer.

The treatment of these cases is easier because the mucous membrane of the vomer is a first-class flap. It has much resistance and does not break; it is very vascular and has no tendency to slough (fig. 13).

Arbuthnot Lane included in his flap a part of white buccal mucous membrane—at least I have this impression. I include nothing but the red nasal mucous membrane of the vomer. Even in the largest clefts this vomerine mucous membrane is always sufficient to reach the nasal mucous membrane on the opposite side.

A buccal flap, as in the preceding cases, completes the closure (fig. 14).

Of course the suture of the soft palate is practised in the same manner as in clefts of the soft palate only.

It is not for me to estimate the intrinsic value of this operation because in order to judge a method correctly it is necessary to take as a criterion successes repeated by different surgeons. Consider what has happened in the case of Brophy's method.

(C) Anatomical Results.

Until now I have operated upon 320 patients. In 1924, at the Surgical Society in Paris,[1]

[1] *Bull. et Mém. Soc. de Chir. de Paris*, April 2, 1924, 497.

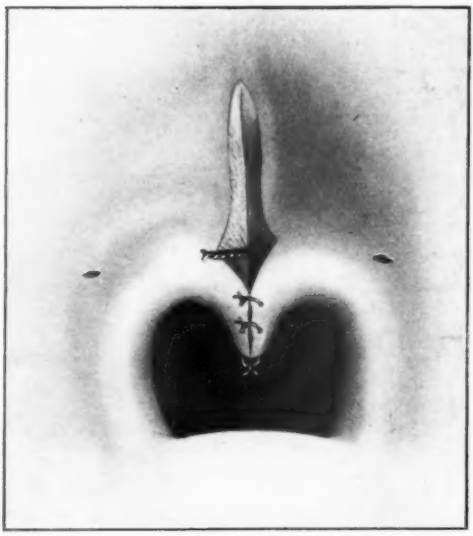

FIG. 9.—The copper thread is tightened. Beginning of the buccal suture.

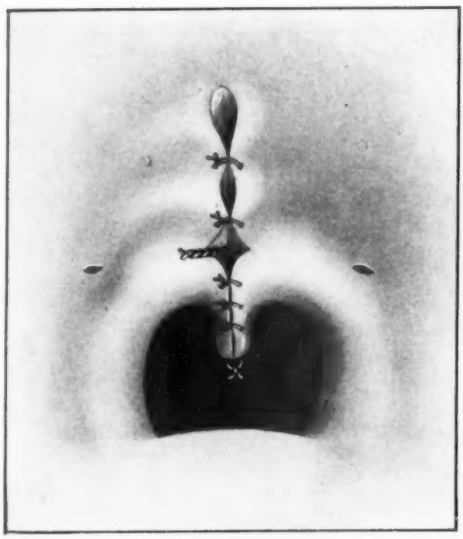

FIG. 10.—End of operation.

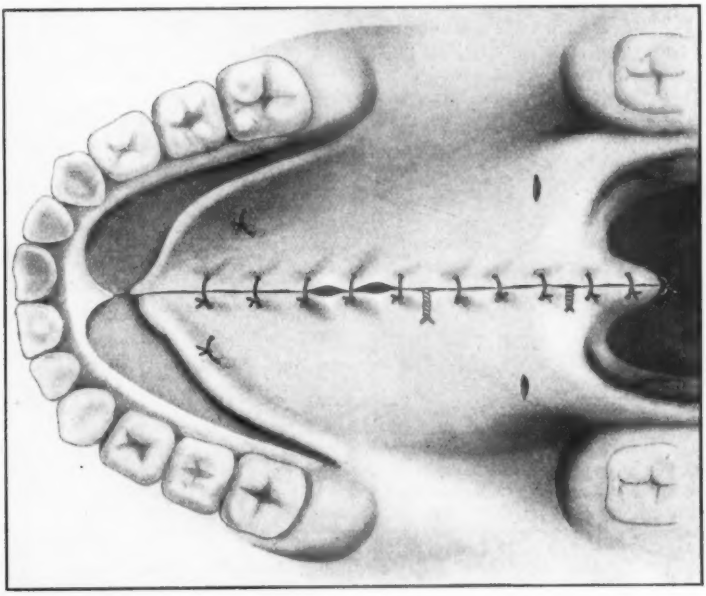

FIG. 12.—Closing the cleft by two flaps, completely mobilized and fixed to the roof.

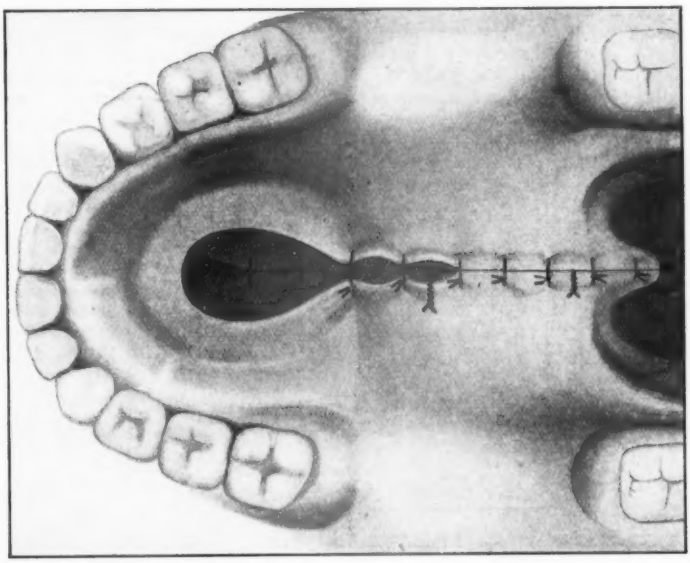

FIG. 11.—Cleft of the soft and bony palate. Impossibility to suture the buccal mucous membrane.

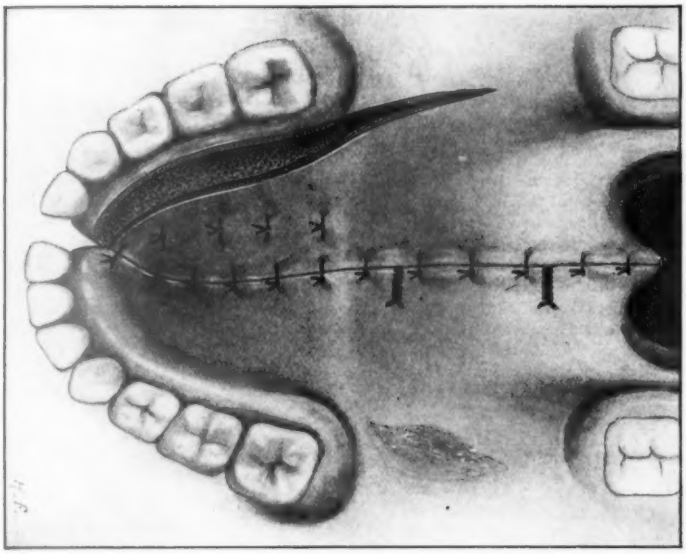

FIG. 14.—Unilateral cleft. Buccal suture.

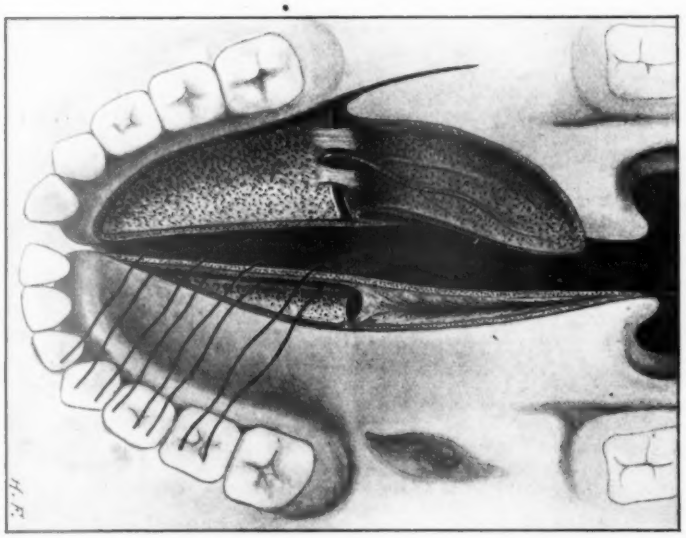

FIG. 13.—Unilateral cleft—flap of the vomerian mucous membrane.

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I published statistics of my first hundred cases out of which I had two deaths:—

		Cure		Failure
	Frequency	One operation	Several operations	Reparable, partly
Total	100 ...	70 per cent. ...	91 per cent. ...	9 per cent.
Soft palate	10 ...	100 ,, ...	— ...	—
Soft and hard palate ...	32 ...	60 ,, ...	94 ,, ...	6 ,,
Unilateral	45 ...	73 ,, ...	89 ,, ...	11 ,,
Bilateral	7 ...	42 ,, ...	86 ,, ...	14 ,,
Already operated upon ...	6 ...	66·6 ,, ...	84 ,, ...	16 ,,

I will briefly recapitulate the special points of my method.

(*a*) The rarity of total non-union—only 2 per cent. Even in two cases in which scarlet fever developed two or three days after the operation, the cleft did not break down.

(*b*) The permanency of mobility of the reconstituted velum. I think this is due to the fact that I do not cut the muscles and above all that I carefully complete the suture of the nasal mucous membrane which ensures rapid healing without scar formation.

(*c*) No slough, no hæmorrhage. This is because I carefully preserve intact the large posterior palatine artery. I have shown[1] that this artery is the only one which supplies the palate.

I recognize that my method is not exempt from criticism. The muscular suture is a delicate operation. The suture of the nasal mucous membrane is sometimes impossible in the front part of the cleft because here it is very thin; a hole may persist between the soft and bony palate.

Shall we ever know how to operate upon a cleft palate with as much security as upon inguinal hernia?

(D) Phonetic Results.

My present technique has produced phonetic results much superior to those obtained when I employed Langenbeck's method. Five children on whom I had operated spoke in the presence of members of the Surgical Society in Paris; their phonation was absolutely normal.

What is the proportion of these fortunate results? This I cannot state with any certainty. My phonetic assistant—Mademoiselle Borel—is preparing a technical work on the subject which will be full of information.

At the present moment this is our impression. Out of ten children operated upon we have two speaking in a manner indistinguishable from the normal, five speaking slightly imperfectly, with nasal intonation of the vowels and defective vocal tone. It is possible for a practised ear to recognize the corrected malformation. Three have defects. The defects are noticeable above all in the occlusives. The constrictives are replaced by guttural sounds. These are the bad cases, in the treatment of which phonetic education has a great value.

The excellence of the result is, of course, not only due to the anatomical perfection of a reconstituted velum, but also to the child's intelligence, and perhaps above all to keenness of hearing. But with a good and very mobile velum the phonation is always better than with immobile and scarred vela; this was my former experience when using Langenbeck's method.

Mr. A. T. Pitts

said that his contribution to this discussion was that of a dental surgeon seeing many cases of cleft palate and being greatly interested in their treatment. It was not without significance that many dentists had been hostile critics of the operative treatment of cleft palate. It might be argued that, as purveyors of a rival method of treatment, their attitude was not wholly disinterested. But though, no doubt, there might be an unconscious bias in favour of prosthetic treatment, the criticism could not be dismissed so lightly. It reflected the undoubted fact that surgery did not always succeed; that even if closure of the cleft were obtained, the functional results might be poor and that a most serious degree of deformity of the jaws might follow. Also that when surgical efforts failed, the original condition was made much worse and the case drifted into the hands of the dentist as a last resort. The criticism of the dentist had some point and was worthy of the surgeon's consideration. For his own part he was not in agreement with such a view; he believed that the best treatment of cleft

[1] *Bull. Soc. Anat. de Paris*, May, 1926.

palate was surgical, though, as at present practised, its results were not ideal. The aims of surgical treatment might be summed up as (1) anatomical closure of the defect with as little disturbance as possible of the normal anatomy of the parts concerned and (2) good functional results as manifested in speech. He proposed to consider the deformity of the teeth and jaws which might follow operation as providing one of the criteria enabling us to compare the merits and demerits of the different operations. It might be stated in general terms that in all cases of complete clefts, whether operated upon or not, there would later on be some irregularity of the incisor teeth due to the disturbance of the tooth band by the failure of development. If the case was operated upon, the effects of the operation would superadd an extra irregularity in the incisor region, but even if not treated surgically some irregularity would exist. Irregularities of the dental arch in the premolar and molar regions were always due to operative interference. If a complete cleft was not operated upon, the dental arch laterally would develop normally.

Four chief methods of treatment had been practised: the Brophy method, the flap operation, the Langenbeck and the Gillies-Fry method. Few cases of the Brophy method had been done in this country. Brophy himself stated that the wires passing through the maxilla could be passed without disturbing the developing teeth, but Blakeway [1], reporting on ten cases done in this country, stated that in some the teeth were exposed during operation and in one case some teeth were shed subsequently. At the period when Brophy advised operation the premolars were not calcified, but the tooth germs were present. No amount of skill could possibly enable the surgeon to ascertain whether or no he was damaging the uncalcified germs of the permanent teeth; only time could show that. In his book Brophy made no reference to the question of any distortion of the dental arches manifest in later life. It was difficult not to believe that they existed. Federspiel [2], in a recently published book on cleft palate remarked that "any injury to the tooth buds at this time (i.e., three months after birth) might not only destroy them, but also displace them from the normal path of eruption. This was especially true in cleft palate cases if an attempt was made to pass wire ligatures through the alveoli according to the Brophy method. The trauma produced by these wires would usually destroy some of these tooth-buds or shift them from the normal position in the mesoblastic tissue." An illustration in Federspiel's book exhibited the result on the dental arch of the Brophy operation. While he (the speaker) was not prepared to say whether this was an average or normal result of the Brophy operation, it was entirely congruous with what one would expect to happen. He felt that the onus was on Brophy to prove that his operation did not distort the dental arch, and in the absence of such proof he considered that he was entitled to assume that not only did Brophy's operation affect the development and position of the permanent teeth, but that the displacement must be severe.

With regard to the Gillies-Fry method it was scarcely possible to offer any criticisms until the results of the operation were available. One might admit that the development of the dental arch would be unaffected, and that the soft palate might be long and movable, but it did not appear to him to be an ideal procedure to condemn the patient from an early age to wear a large plate covering the deficiency of the hard palate. Children were very self-conscious of any apparatus especially when they went to school. The psychological effect on their mental development needed to be considered, and he was sure that from the point of view of their physical and moral comfort, surgical treatment was better than a prosthetic appliance. Such an appliance was not easy to retain, especially if the dental arch was complete. Later on in life the fact that the patient's well-being depended on the integrity of a plate which, if broken or lost, might not be easily replaced, might be a serious economic handicap.

The two operations which have been performed most often in this country were the flap operation popularized by Arbuthnot Lane, and the Langenbeck operation. In a paper he (the speaker) wrote in 1922 [3], he pointed out that both these operations tended to produce some distortion of the dental arch, but that the type of irregularity produced differed in each operation. The flap operation tended to bring about a unilateral narrowing of the arch on the site from which the flap is taken (figs. 1, 2, 3). The Langenbeck operation tended to produce a bilateral narrowing (figs. 4, 5). In the majority of cases which had been operated on it was possible by looking at the palate to say which operation had been done. He had seen a large number of palates operated on by both methods, and had no hesitation in saying that the deformity produced by the Langenbeck method was far less than that following the flap operation. For several years he had had the opportunity at the Hospital for Sick Children,

FIG. 1.

FIG. 2.

FIG. 3.

Great Ormond Street, of seeing the results of Langenbeck operations in which the narrowing of the dental arch was extremely slight—far less than the least marked results of the flap operation. He had shown elsewhere [4] that the contraction of the dental arches could be improved by orthodontic treatment, though such treatment was prolonged and difficult. From this point of view the bilateral contraction produced by the Langenbeck method was more amenable to orthodontic treatment than the unilateral contraction caused by the flap operation.

In attempting to decide on the best method of treating cleft palate it was necessary to consider all the available criteria. The ultimate result on the teeth and jaws would not in itself afford a sufficient justification for stating that the Langenbeck operation was the best; this criterion of success needed to be considered in common with other factors. He submitted

FIG. 4.

FIG. 5.

that in the past it had not received enough weight. If it could be shown, for instance, that the percentage of success in closing the cleft was greater in the flap than in the Langenbeck method or that the functional results on speech and deglutition were better, then the greater degree of deformity produced on the dental arch might not outweigh the first two advantages. But if operative success and functional results in the two operations were equal, then the effect on the dental arch might well be the determining factor in deciding which operation was the better. As a dentist he did not pretend to be in a position to judge all the criteria on which a final decision could be based but the impression he had gained was that so far as closure of the cleft and the results on speech were concerned, the flap operation had no advantages over the Langenbeck operation which would outweigh its severe effects on the teeth and jaws.

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Another point he would like to make was the remarkable discrepancy which often existed between the anatomical and functional results. The dentist who saw post-operative results at a late stage was favourably placed for noting the ultimate results of operation. One would imagine on *a priori* grounds that the greater the approximation to the normal the better would be the functional results. But this was not always so. One often saw cases in which the anatomical results of operation were excellent and yet speech was poor. Conversely, one often saw cases in which the soft palate was short, moved little and could not touch the posterior wall of the pharynx; there might be defects of the hard palate in addition and yet speech was excellent. How could these diverse results be correlated and explained?

Doubtless the mentality of the child and its capacity for being educated, as well as careful training in phonation, played a part, but he questioned whether they were a sufficient explanation. It would seem that there were other factors concerning which we knew little which accounted for such good functional results when the surgical restoration was obviously incomplete, and vice versa. In view of this not uncommon discrepancy between form and function, the argument of Gillies and Fry that the short soft palate produced by the Langenbeck and flap operations always led to loss of function and diminished the value of such operations, lost some of its force.

The third point he would make concerned subsidiary operations when the primary operation on the palate was wholly or partly a failure. He was sure that the prospects of success diminished with each successive operation. He had seen many cases in which as many as ten or more operations had been performed and finally the surgeon had referred the case to the dental department. In such cases dental treatment was made much more difficult; its chance of success was greatly lessened and sometimes became impossible. He doubted whether repeated attempts at surgical treatment were wise. The analogy of the horse in the jumping ring which was turned out after the third refusal might be appropriate. Perhaps the surgeon would often be wise to abdicate early in favour of dental treatment before the tissues had become so damaged and distorted that prosthetic restoration became impossible or only imperfectly successful. The temptation to attempt closure of small residual holes left in the palate was naturally great. But in his experience even considerable holes might not have any effect on speech. The tendency was for them to become progressively less. Furthermore, since a denture was nearly always necessary in complete clefts in order to restore the deficiency of incisor teeth, a small hole in the hard palate was easily covered by a plate. There was one supplemental operation which was often useful, namely, an epithelial inlay to deepen the lip sulcus. In many cases of complete clefts associated with harelip, the lip sulcus was partly obliterated by operation. This immobilized the lip and tended to give the face a wooden expression. It interfered with speech and made prosthetic restoration more difficult. He had had the opportunity of doing a considerable number of epithelial inlays with his colleagues at the Hospital for Sick Children. The results had been invariably successful, and were a great improvement.

In conclusion, he would urge the necessity of a still greater liaison between surgeons and dentists in the treatment of cleft palate. Surgery and dentistry should not be regarded as rival methods of treatment but as complementary and equally necessary to promote final success. The last decade had seen a far closer *rapprochement* between surgeons and dentists, with beneficial results; but a still closer relationship was possible and desirable. The ideal treatment of cleft palate involved not only the perfection of surgical technique but also efficient pre- and post-operative treatment extending over a long period. Its successful accomplishment demanded the closest co-operation between the various members of the healing craft concerned in the treatment of the different aspects of this condition.

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Mr. CYRIL A. R. NITCH

recorded eighty-six cases, of which forty-three had been traced. The post-operative mortality was nearly 5 per cent.

His cases were divided into three groups according to the type of operation performed.

(I) *Flap Operation of Lane.*

Number of cases 23. Died 1 (diarrhœa and vomiting)

Partial clefts ...	15	...	Held	14	...	With a hole	7	...	Gaped completely		1
Complete clefts ...	8	...	,,	7	...	—		...	,,	,,	1
Total ...	23			21 (91 per cent.)			7				2

The hole in these cases was small and situated at the junction of the hard and soft palates.

Six cases were traced and in all the soft palate was both short and rigid. Speech was poor in three and bad in three.

(II) *Langenbeck* (with or without small lateral incisions).

Number of cases 12 ... Held 5 (41 per cent.) ... Hole[1] 5 ... Gaped completely 2

[1] The hole was so large that repair required an extensive plastic operation.

One case was traced and in this the speech was poor.

The cases in groups 1 and 2 were all operated upon before 1913. Subsequently, a modified Langenbeck operation with long lateral incisions, transfixion wires and metal plates for relief of tension, was performed with the result that anatomical failures were reduced from 50 per cent. (Langenbeck) to 17 per cent. (modified Langenbeck).

(III) *Modified Langenbeck.*

Number of cases 51. Deaths 3.

Partial clefts ...	35	...	Held	30	...	Hole[1]	7	...	Gaped completely		5
Complete clefts ...	16	...	,,	12	...	,,	3	...	,,	,,	4
Total ...	51			42 (82 per cent.)			10				9 (17 per cent.)

[1] Successfully closed clefts in which a *small* hole developed in some part of the suture line, which either closed spontaneously, or was too small to cause any inconvenience or disability, or was easily closed by a small flap.

Four of the successful cases had been operated on one or more times by others. Seven of the nine failures were operated on again successfully.

In thirty-five of the above cases which were traced, speech was:—

Good in 13, i.e., perfect or nearly so.
Poor in 17, i.e., typical cleft palate speech.
Bad in 5, i.e., practically unintelligible.

He (the speaker) considered 1½ to 2 years the optimum age for operation so that speech training could be commenced before the child had learned to talk badly. For wide cleft, operation was deferred until the palatal processes had grown sufficiently to ensure a reasonable chance of success.

An attempt was always made to close the whole cleft at one operation, provided the anterior palatine artery could be preserved. Failing this, the anterior end of the cleft was left unclosed.

Cleft lip and prominent premaxilla should be rectified at the age of three months.

Pre-operative Treatment.

The patient was kept in hospital for a week before operation to enable the child to become accustomed to nurses and surroundings. The mouth was syringed with an alkaline lotion after each meal. If tolerated, this was continued after operation, if not, a drink of cold water was given.

The teeth and tonsils were attended to, but adenoids were removed as they helped to fill up the naso-pharynx.

Operation.

Lateral incisions were made in all cases, and in many they extended nearly to the last lower molar tooth. Their length was so adjusted as to permit the edges of the cleft to come together without tension. Immediately behind the hard palate all the structures of the soft palate were divided by the lateral incision, but, inferiorly, often only the mucous membrane was divided. Tension wires transfixing the soft palate and lateral metal splints were always used. Coaption sutures were of silk.

Before the child left the hospital, the mother was instructed to massage the soft palate daily, since good speech depended mainly on mobility of the soft palate.

In cases with complete clefts and consequent deformity of the nasal cavities, the patients seldom spoke as well as those with a partial cleft, no matter how good the anatomical operative result.

Adequate and often prolonged speech training was necessary in nearly every case.

Mr. Cortlandt MacMahon

discussed the speech training of the cleft-palate patient. In many cases the letters "K, G, D, T," were entirely missing after repair of the palate, and the letter "S" was a severe blemish in the speech. To bring into being the "K" the tip of the tongue should be pressed downwards and gently backwards behind the lower teeth by the person who is to educate the speech of the child. This action would cause the back of the tongue to come into contact with the soft palate, and if the patient then tried to make the sound of "K" this would probably occur immediately; in any case it should occur within a few days or weeks. "G" having the same contact as "K" between the back of the tongue and the soft palate, would occur if the letter was helped by making the sound of "N" in the nasal cavities and then continuing the "N" to the sound of "NG" which was the true sound of "G." "D" must be produced with the tongue on the alveolar margin behind the top teeth and it was easily produced if it was helped by the sound of "N." The letter "T" would occur after "D" had been established. It had the same tongue contact, but it was often a rather difficult letter to establish. The letter "S" was always a very difficult one to deal with. The back of the tongue was high as a result of the repair to the soft palate and this caused the tip of the tongue to lie flat against the lower teeth. Its normal position was immediately behind the top teeth. By continually depressing the back of the tongue on the sound of "Ah" the back of the tongue would be made to sink definitely in the throat and the tip of the tongue would then easily accommodate itself on the alveolar margin behind the top teeth. The back of the tongue and the soft palate moved in sympathy, and the lower the position of the back of the tongue the more effort would be made by the levator muscles to raise the soft palate. The floor of the mouth and the back of the tongue were low when the voice was deep and this fact would compensate to a considerable degree for the immobility of the palate, if immobility was present, and the air would be much less likely to pass into the nasal cavities during speech. With a freely-moving soft palate the letter "S" should be produced absolutely normally if the speech had been trained very soon after operation.

The flap operation often gave good speech results if the patient received proper training, and training in the flap operation was very necessary. The Langenbeck method of operation often gave automatically good speech and the palate moved more freely than in the flap operation, as there was no scar tissue in the latter operation and the elasticity of the palate was greater from that fact.

Mr. MacMahon referred to a reprint of an article on the cleft palate speech which he published in the *British Dental Journal*, September 15, 1920. If the instructions in that article were carried out by the mother or nurse of the little patient, further instruction would often be unnecessary.

In conclusion he said that it was most essential that the training of the speech should be begun very early after the operation. Depressing the back of the tongue on the sound of "Ah" 100 times daily would ensure the best possible movement of the soft palate, and enable the tongue to get its proper contacts with the hard and soft palates, and it was always most essential that the pitch of the voice of the cleft-palate patient should be low.

Mr. H. D. Gillies.

In regard to secondary operations on the palate, there are just two points which it may be of service to bring to the notice of this meeting.

Both have been found to apply in a fair proportion of cases met with in my experience.

The first point is in connexion with *the position* of the soft palate. This is often found in a position so far forward that it cannot take its share in the closure of the oro-nasal passage, and, in consequence, the worst type of voice is produced. When such a palate is immobile and scarred or atrophied, conditions for speech are at their worst.

The second point is in connexion with the position of the upper lip and nose in relation to the face in general. These portions of the face are situated too far back, as a result of the approximation of the maxillæ which has been produced by the surgical closure. This deformity is at its worst in cases in which the premaxilla has been removed without adequate dental provision for its substitution.

In regard to the soft palate,[1] the condition of the great majority of these cases will be vastly improved (*a*) by the severance of the soft from the hard palate; (*b*) by the provision for an epithelial covering to all raw areas; (*c*) by the interposition of an appliance between the soft and the hard palate. In this way the soft palate with its muscles will be placed in such a position that it can assist in closing off the nasal from the buccal cavity. The speaker has done this operation in a considerable number of cases with very marked improvement of speech in all.

In regard to the posteriorly situated lip and nose, the most satisfactory remedy appears to be the use of what is called a "buccal inlay,"[2] by means of which the soft tissues are separated from the hard, raw surfaces, and are skin-grafted, a dental plate being afterwards fitted, carrying the requisite contour and having teeth occluding with the lower.

When the teeth in the upper jaw have been carried in to a very marked degree these seriously interfere with the mobility of the tongue and the power of speech. After consultation with the dental surgeon some or all of these teeth may be removed, or they may be covered in by the dental plate.

Mr. C. H. FAGGE

said that up till the end of 1924 he had operated upon 114 cleft palates and on an analysis of these cases his remarks would be based; he was indebted to his Surgical Registrar for a most careful review during the past twelve months of his old cases.

Palatal clefts could be divided into: (1) those involving the soft palate; (2*a*) the soft with a small amount of hard; (2*b*) the soft and hard as far as the anterior palatine foramen; (3*a*) complete cleft palate and unilateral hare-lip usually on the left side, and (3*b*) complete cleft palate and double hare-lip. In his opinion a Langenbeck operation was entirely suitable for the first two and a large majority of the third class (2*b*); whenever he had met with a wide cleft in this class he had closed it by a Lane's flap operation, but in his experience difficult clefts of this sort were very rare indeed. Of this series a complete cleft with a unilateral hare-lip was by far the commonest type, amounting to more than one-third of the total (forty-five cases); for this he had in former years carried out a complete Lane's operation on the palate both hard and soft, but as he found this too severe an operation for small babies and because of the tendency of the soft palate to break down, he had abandoned it for a Lane flap operation on the hard palate, followed later by a Langenbeck operation on the soft palate; the hare-lip was dealt with at the first operation. Complete cleft palates with a double hare-lip were treated on exactly the same lines, i.e., at the first operation the hard palate was closed with Lane's flaps and the lip was sutured, the soft palate being closed by a Langenbeck operation about three months later.

Whatever other speakers might think personally about the most suitable date at which cleft palate operations should be performed, it was obvious to those who had taken part in the debate sixteen years ago that the majority of operators, like the opener of the discussion, were now operating at a much earlier age than was their custom sixteen years ago; this was attributable to the influence of Lane's teaching. For his own part, he (the speaker) had experimented, largely owing to circumstances beyond his control, on the question of the best age for the operation and had convinced himself that in his hands and by the method which he employed, the most suitable age for the first operation was during the second or third month of life, so as to allow the second operation to be completed before the troubles of first dentition began. After the performance of a certain number of operations of one kind, everyone should know his own chances of success and his personal limitations; he (the speaker) knew that he could insure a very reasonable anatomical success by operations in young babies; the cases which puzzled him and often defeated him were those in which he had no chance of operating until the baby was two or three years old. He would like to emphasize the fact that he had not met with a cleft of the hard palate which he had been unable to close by Lane's method,

1 *Brit. Med. Journ.*, March 5, 1921 (Gillies and Fry).
2 *Idem.*

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nor was there among his series a single case in which the hard palate had broken down. He did not find that closure of the hard palate by a flap made the second operation on the soft palate more difficult. It must be admitted as demonstrated by some excellent casts which Mr. Skinner had made from some of the cases, that narrowing of the anterior arch occurred on the side on which the flap was raised and turned inwards. He was not convinced that this did not occur without any operation and it was certainly fairly common after the Langenbeck operation as demonstrated by the drawings shown by the opener of the discussion. Personally he regarded this defect as a comparatively small one and was in complete agreement with those who emphasized the supreme importance of a functional soft palate over every other consideration.

With regard to results, there had been eight deaths within a few hours and six within three weeks, giving an operative mortality of 12 per cent.; in twelve cases the patients had died subsequently from various causes unconnected with their palates. His Surgical Registrar had collated results according to the type of operation performed; most of the information had been gained by seeing the patients, but in some cases the result was reported, usually by the patient's medical attendant. For what they were worth he would state briefly his "reported" results.

Langenbeck.

Number of cases 38 [1]
Number of operations 63 [2]

Number of "reported" successes 23 (60·5 per cent.)
Number of partial successes 10 (10·5 per cent.)
Number of failures 2 (5·2 per cent.)
In 83 per cent. the hard palate was satisfactorily closed

Combined Operation.

Number of cases 35 (31 under 1 year)
Number of operations 89 [2]
Mortality within 5 hours *nil*
Mortality within three weeks 2 (5·7 per cent.)
Number of "reported" successes 22 (62·8 per cent.)
Number of partial successes 10 (22 per cent.)

Types: 3*b*—8; 3*a*—25; 2—2

In 20 per cent. the upper dental arch was well developed and occlusion good. The common error was an in-drawing of the alveolar arch in the region of the incisors, canines and premolars of the affected side with irregular eruption of those teeth. In 60 per cent. the speech was good and fair in the remainder.

Lane.

Number of cases 18 (17 under 1 year)
Number of operations 19
Mortality within 5 hours 2 (22 per cent.)
Mortality within 3 weeks 2 (22 per cent.)
Number of "reported" successes 3 (33 per cent.)
Number of "reported" failures *nil*

Types: 3*b*—7; 3*a*—7; 2*b*—1; 2*a*—1. Unclassified 3

With regard to Sir James Berry's remarks on the use of tension sutures he (the speaker) was in complete disagreement with this practice. In his view, if such sutures were under tension they sloughed out and if there was no tension they acted as unnecessary foreign bodies. In the performance of the Langenbeck operation his one principle was the avoidance of tension: the pared cleft was sutured with interrupted stitches of the very finest gossamer gut; lateral incisions were made in almost every case, and usually they passed back through the anterior pillar of the fauces: he tried to preserve the descending palatine artery, but its division was not infrequent and had no influence upon the healing of the cleft. He had given up any form of local after-treatment except the gentle cleansing of the mouth with sterile water after

1 To these should probably be added twenty-three cases of which the hospital notes give no details as to the exact operation employed.

2 The large number of operations as compared with the number of cases is misleading; it includes all operations on the hare-lips and secondary operations for the closure of small holes at the junction of the hard and soft palates. It was soon realized that many of these holes healed spontaneously and secondary operations were accordingly discontinued.

each feed. In his opinion the best post-operative sedative was brandy. Before leaving the hospital the child was put under the care of a dental surgeon, and steps were taken to ensure that as soon as it began to speak, it came under the care of a proper teacher; this did more to produce a good functional result than the most perfect operation.

Mr. W. KELSEY FRY

said: I am speaking from the aspect of a dental surgeon who is particularly interested in cleft palates. I have been associated with my colleague, Mr. Gillies, in emphasizing the importance of a functional soft palate for procuring good speech, and it has been pleasant to hear this point emphasized so much by the previous speakers. I was delighted to see so many successful cases from treatment by the various types of operation, because, in my position as Director in charge of the Prosthetic Department of Guy's Hospital, I come into contact with many cases which have proved unsuccessful from operative treatment. I have now under my care about 300 patients, all of whom present the characteristic features of operative failures, i.e., the speech is bad from absence or shortness of the soft palate, contracted dental arches due to scar tissue in the palate, and a falling back of the upper lip due to either removal of the premaxilla or its drawing back towards the middle of the palate. These cases are most difficult to treat from the prosthetic point of view, and success in speech can only be obtained by the use of intricate and cumbersome apparatus. I am fully conscious that with clefts involving the soft palate only, or perhaps part of the hard palate, the ordinary operations advocated should give satisfactory results, but I am more concerned with complete clefts. In advising on treatment at birth it is necessary to concentrate on attaining the four cardinal results required, viz., early nourishment, good speech, good occlusion of the teeth, and good appearance. In order to ensure good early nourishment the infant is fitted with a small sucking plate at birth, which is made by taking an impression of the mouth and constructing a small plate in vulcanite. This plate immediately relieves the difficulty of feeding, as the infant can be fed in the usual way with a bottle. There are times even when they can take the breast, but this depends entirely upon the supply of milk. The plate is only worn for feeding purposes, and at the end of seven or eight months is gradually discarded as the child will have learnt to use the bottle without it.

The speech of the child has now to be considered, and whether you should aim at closing the whole of the hard and soft palates—and so risk obtaining a short and functionless soft palate—or whether you would not be more certain of obtaining good speech by "putting all your eggs into one basket" and being quite sure of making a freely movable and long soft palate. In treating cases in this way you will of course leave a large hole in the hard palate, but this can be closed perfectly easily by a simple vulcanite plate, such as that constructed by any dental surgeon, and similar to those worn by many people. Mr. Pitts has rather argued against leaving a hole in the hard palate on the grounds that it necessitates the patients wearing a plate, but almost in his next sentence he said (if I remember correctly) that owing to the absence of one or more of the front teeth these patients had to have a small plate constructed. That is exactly my point—patients with complete clefts do invariably have to wear a plate to replace one or more of the front teeth, and such a plate needs to be extended over the hard palate, whether the surgeon has closed it or not; therefore, why use what I may term precious tissue to close the hard palate when, for the all-important function of speech, such tissue would be invaluable for the formation of the soft palate? On behalf of the hundreds of little mutilated patients it has been my misfortune to have under my care, I do earnestly ask you to consider this point, and so, in my opinion prevent further cases coming under my care. I am not here to-night to advocate any special surgical technique, because I am not a surgeon, but I do say to you that you should first only reconstruct the soft palate, then later, if you think you can close the hard palate without bringing the soft palate even a millimetre forwards, by all means do so. If I were a patient I should require a guarantee that no pulling forward of the soft palate took place during the operation of closing the hard palate.

The questions of occlusion of the teeth, and of appearance, are again concerned with the closing of the hard palate. When the hard palate is not closed the maxillary teeth remain in good occlusion with the lower teeth, and the contour of the face is maintained; but when the hard palate is closed and scar tissue is formed, the two parts of the maxilla are approximated by the scar tissue and the occlusion of the teeth and the contour of the face are destroyed.

To summarize: the closure of the hard palate may be the cause of a contracted arch, a loss of contour of the face, and bad speech, by the pulling forward of the soft palate towards the middle of the palate; all of these could be avoided by the use of a plate to close over the opening. Plenty of tissue to form a freely movable soft palate will be left: this will restore to the child the all-important function of speech.

Mr. F. W. GOYDER (Bradford)

said that his colleagues did not touch these cases, and therefore he had little opportunity of comparing his results with those of others. He had dealt with 150 cases, but was unable to give exact statistics as to the ultimate results regarding speech. He began by performing Lane's operation in early infancy, but soon abandoned wide flaps. Operation by the use of a restricted flap at any age, in his hands, was a reliable method of closing the cleft, but the speech results were bad. He now seldom attempted to close the cleft in a child younger than 3 years of age. Since the war he had performed Langenbeck's operation with increasing frequency, but with less certainty of closing the cleft than when a flap was used. In his last twenty-two cases he had performed Langenbeck's operation eleven times. When the clefts were very wide he had used a flap in order to avoid long lateral incisions, which left larger raw surfaces than a restricted flap operation. After hearing Mr. Addison's remarks and seeing his cases, he felt inclined to try Mr. Addison's method. In none of his own cases could he say that speech was perfect, and he felt strongly that, when no training was given after cleft palate operations, good speech never occurred.

Mr. L. E. BARRINGTON-WARD

said his contribution to this debate was based on a study of 148 cases of cleft palate upon which he had operated since April, 1914. He had kept complete records of every case, and had re-examined all cases as far as possible at yearly intervals.

Of these, 132 were operated upon at the Hospital for Sick Children and sixteen in private practice.

Three patients died as a result of the operation (two hospital cases and one private). One died from reactionary hæmorrhage which was not recognized or treated in time; one died on the table from status lymphaticus; and one died suddenly some hours after operation from some unexplained cause. He had treated 111 cases without a death before these fatalities occurred, and he looked upon these deaths as a series of misfortunes which might have been avoided.

He had had one case of complete failure. It was a horse-shoe type of cleft of the hard and soft palates with very little tissue, and he was perhaps unwise to attempt repair. No further operative treatment was contemplated.

In two cases the alveolus was still cleft, and he had advised prosthetic treatment instead of further operation.

In three cases there was still a deficiency in the hard or soft palate.

In five cases the patients were awaiting further operation.

Although he had taken particular pains to keep in touch with all his cases he had lost sight of twenty-nine. Of these, fifteen patients still had some deficiency in the palate when last seen. It was reasonable to assume that as they were cases of central deficiencies of small extent, in some of them there might be complete healing. It was well known that there was a spontaneous tendency to closure of these small central defects.

Of these 148 cases, eighty-one were complete clefts, ten associated with a double hare-lip, seventy-one with a single hare-lip; fifty-one involved the soft and part of the hard palates only, and sixteen were clefts of the soft palate. Sixteen had been previously operated upon elsewhere unsuccessfully.

The estimation of the speech was a difficult matter, and the operator was perhaps an indifferent judge. He had endeavoured to set himself a high standard, and had classified speech as "good," "fair" and "bad." By "good" was meant clear speech with no cleft palate intonation. By "fair" was meant clear, intelligible speech, yet with the characteristic intonation associated with cleft palate. By "bad" was understood the remaining conditions. During the last two years he had examined fifty-eight cases of an age suitable for a fair test, and he had found that the speech was good in twenty patients, fair in eighteen, and bad in twenty, of whom four were mentally deficient.

As regarded the number of operations necessary to achieve these results: 105 patients were operated upon once, thirty-one were operated upon twice, six were operated upon three times, and three were operated upon four times.

CONCLUSIONS.

These could be briefly stated, as follows:—

(1) In regard to the age at which the operation should be performed in the case of cleft palate associated with a hare-lip, the lip operation should be undertaken at the age of 4 weeks. Union of the lip had a profound effect on the palate, as it drew the alveolar margins together and caused retrogression in the overgrown septum. If the lip operation was carried out much later, the alveolar margin would be irregular. Operation on the palate should not be undertaken until the child was from 18 months to 2 years of age in the case of complete clefts. If done even later no harm would result. In his early cases he used to operate on the children at 1 year or even 6 months of age, but experience had proved that a late operation had no ill-effects as regarded either the anatomical or the functional result. Clefts of the hard and soft palates could always be closed at 18 months, clefts of the soft palate alone at 12 months.

(2) The operation performed in this series of cases had been the Langenbeck operation of median suture.

(3) He attempted to close the whole palate at one operation, with the mental reservation that a secondary operation might be necessary. In 105 cases he had only had to operate once.

(4) As regarded lateral incisions he made no exact rule. Each case must be taken on its merits, as much depended on the type of deficiency. The idea was the relief of tension, and no more and no less should be aimed at. If the gap was narrow a very short lateral incision will be sufficient. If the gap was wide a long incision would be necessary. He paid more attention to separating the attachments of the soft palate very freely from the back of the hard, and he had found partial division of the palato-pharyngeal fold of great value in relieving tension at the widest part, viz., just behind the hard palate.

(5) Adequate preliminary treatment was of paramount importance. If the tonsils were septic they should be dissected out. If the teeth were decayed they should be removed. It was of great value to accustom the child to regular spraying of the mouth before operation. He instructed the mothers to carry this out, so that the child became thoroughly familiarized with the procedure and did not resent such treatment after operation.

(6) *Sutures.*—He used interrupted sutures of ophthalmic silkworm gut, inserted with a Lane's needle-holder and a Lane's needle. Great care was taken to produce exact apposition. He believed that relaxation sutures were harmful, for in his experience they frequently cut out and in any case the palate should be so freed that they were unnecessary.

(7) Dental treatment was most important. The aid of the dental surgeon should be invoked before operation. After operation his help was invaluable in regulating irregularities of the alveolar border. In cases associated with a double harelip with the soft tissues of the lip adherent to the premaxilla, an epithelial inlay produced a mobile lip and improved not only the appearance but also the speech.

(8) *Speech Training.*—The question of speech perplexed him greatly. In this series of cases he could find no definite relationship between a perfect anatomical and a perfect physiological result. He had cases with the most perfect soft palates in which patients spoke exceedingly badly, and he had also cases where there were short, scarred, indifferent soft palates in which the patients spoke remarkably well. Speech was the result of cerebration, and he felt sure that cleft palate speech had more to do with the higher centres than the actual anatomical appearance of the palate. He agreed that every effort should be directed to reproduce the normal as far as possible, but he would lay more stress on speech training than on any other factor. At the Hospital for Sick Children, Great Ormond Street, they had a speech clinic for the specialized teaching of their patients after operation, and it had proved its value. The speech of the child depended first on the intelligence of the child, and, secondly, on the intelligence of the mother. In many hospital cases the children were of feeble intellect and their chances were small. In giving a prognosis on the question of speech to any parent, he would be guided more by the intelligence of the parent than by the nature and width of the cleft in the child.

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Mr. T. T. Higgins

said that as his own group of cases only numbered forty-seven—all operated on since 1921—he was not able to judge of the ultimate results, especially with regard to the all-important questions of articulation. He felt stimulated, however, to state his personal experience of the "Gillies-Fry" operation and of the value of the long lateral incisions as employed by Addison.

When Gillies and Fry first put forward their suggestion he had adopted it in six cases of varying types of cleft. He had found it easy to obtain a satisfactory closure, the only difficulty being to cover completely the raw area at the anterior end of the soft palate. For this purpose he had utilized the parings from the side of the cleft, but these were not always long enough. An unsatisfactory graft meant a scar, which subsequently contracted and shortened the palate.

He had been pleased with the soft palate which resulted from this operation. It was long and mobile as in the case shown. The chief drawback in his experience had been the difficulty of fitting the child with a dental plate to close the defect in the hard palate. His dental colleague, Mr. Pitts, who was most interested in and helpful in these cases, did not consider it feasible to fit such plates until the age of 6 or 7 years.

As the operations were performed round about two years of age, this left the child, during the years it was learning to talk, with an open hard palate—obviously a serious drawback. Nevertheless the speech of these children already promised to be quite good. Time only would show whether articulation in these cases was markedly better than in those operated upon by more orthodox methods. This was the criterion by which they must be ultimately judged.

Latterly, in carrying out the side-to-side closure by the Langenbeck-Fergusson-Berry method, he had been employing the long-lateral incisions advocated by Addison. He had followed his technique very carefully and had removed the tonsils and adenoids in all cases himself. The tonsils were dissected gently and all mucous membrane was preserved so as to minimize the subsequent scarring. He had found the tonsils obviously septic in almost every case and in several they had actually contained pus.

He felt convinced of the truth of the assertion that the tonsil was a dangerous focus of infection. With regard to adenoids, he always removed them too. They must share the tonsillar infection if this was present, and he could never believe they could assist speech very much since they were to atrophy in later childhood. He had operated on fifteen cases in this way and in fourteen of them had achieved perfect anatomical closure of the cleft throughout its length—this by one operation only. He had certainly not succeeded in obtaining anything like satisfactory results without the use of the long lateral incisions. These incisions healed rapidly, the scars were not noticeable, and he did not believe that they would ultimately interfere with the mobility of the palate, because in making them correctly there should be no extensive division of muscle fibres.

Mr. W. E. M. Wardill (Newcastle-upon-Tyne).

For many years past, the treatment of cleft palate has shown very little improvement except with regard to standardization of technique in the various operations. Different methods are practised by individuals with fairly uniform results in that approximation of the parts in the middle line is almost perfect. The frequent discussions on the subject, however, indicated an attempt on the part of surgeons to justify their various procedures and surely point to entire dissatisfaction with the functional, as apart from the anatomical, results. That such dissatisfaction is warranted is evident from even a cursory examination of any large series of cases after operation. It is true that most of the patients do not tend to regurgitate food through the nose, and that naso-pharyngeal hygiene is raised to a higher level. Bad speech, the greatest disability from which the patient suffers, remains. A few fortunate cases are benefited in this direction; the large majority have to depend upon the efforts of the elocutionist who teaches them certain pharyngeal and lingual gymnastic tricks, usually to their great benefit, but only in rare cases is normal speech restored.

This state of affairs merits a critical inquiry into the anatomy of speech, both in the normal and in the sufferer from cleft palate.

ANATOMY OF SPEECH.

It is necessary to draw attention to the function of the naso-pharyngeal valve, which during normal speech is constantly kept at work. An analysis of one's own actions will speedily convince anyone of the mercurial mobility of this valve. For instance, during the pronunciation of the letters "S" and "L," the valve is closed and air is prevented from passing into the nose; in pronouncing "N," however, nasal resonance is produced only if this passage remains open. When the three letters are combined, as in *snail*, a rapid opening (N) follows complete closure (S) and is again followed by closure (L) in finishing the word. Examination of ordinary language shows that the valve is more frequently used in the closed position than in the open, the only sounds absolutely requiring the open position being the M and the N. This fact will be readily appreciated by anyone who has suffered from "a cold id by dose" (a cold in the nose). Much more disastrous effects upon speech are observed when the pharyngeal valve remains permanently open as in cleft palate. Speech is then distinct or otherwise according to the relative proportions of sound projected through the lips and the nose. As before mentioned lessons in elocution are able to make the subject of cleft palate understandable, but no amount of training can ever replace the incompetent or lost pharyngeal valve.

What, exactly, is the anatomy of this valve? Roughly it consists of two parts, a pharyngeal and a palatal. The pharyngeal half is formed by some of the transverse fibres in the upper part of the constrictor pharyngis superioris, which, when contracted, produce a well-marked transverse ridge behind, meeting the posterior surface of the soft palate in front. The existence of this ridge and the free mobility of the superior constrictor are obvious in any case of cleft palate in which no operation has been done. The palatal part is formed by the soft palate, movement of which is effected through two opposed groups of muscles. The main adductors are formed by the levators of the palate which draw the latter upwards and backwards and at the same time narrow the naso-pharynx laterally. The adductors are the palato-pharyngeus, palato-glossus, and the tensors of the palate.

Normally the palate hangs downward as a long veil-like structure which is not far separated from the posterior pharyngeal wall. The chink so formed, although narrow antero-posteriorly, is nevertheless wide from side to side, the consequent air space reaching a fairly large total. From this it follows that small movements measured in terms of linear dimensions allow of wide variations in the cross-section area of the pharyngeal airway, and in this respect the valve bears some general resemblance to the valves in a motor-car engine.

Critical inquiry into the actions of the two parts of the pharyngeal valve during speech discloses the fact that definite information is unobtainable, and one is left to draw conclusions very often from mere surmise. My conclusions are based upon analysis of the actions of my own naso-pharynx and on deductions from the anatomical arrangements of muscles. The following would seem to be the facts of the case. The soft palate is highly mobile and the activities of its muscles are responsible for the rapid variations in the state of the naso-pharyngeal airway. The superior constrictor and its transverse ridge remain in a state of contraction so long as speech is being emitted; its function is that of forming a buffer against which the soft palate can flap. The structure of this muscle is such that one would hardly expect rapid and free movement; its under-surface is in connexion with loose connective tissue, and, were free movement its function, a bursa might be expected in place of this. Again, the superior constrictor acts more slowly than most voluntary muscles; it appears to contract slowly and relax slowly. If those present are at all conscious of their own muscular movements, they will readily recognize the feeling of slow relaxation of the posterior wall of the pharynx every time they cease to speak. Summarizing, then, normal speech depends upon the contraction of the superior constrictor which produces a "seating" for the "flap-valve-like" action of the soft palate.

CAUSES OF CLEFT-PALATE SPEECH.

Although the main principles underlying the foregoing remarks are generally accepted, most surgeons seem to be agreed that cleft-palate speech is not entirely due to anatomical loss of this valve. There is no doubt that this is partly true, because, in cleft palate, however carefully the defect has been repaired, anatomical deformity still remains and it might be expected that this would be associated in some degree with functional derangement. On

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the other hand, many persons in possession of a perfectly normal speech mechanism can imitate almost to perfection cleft-palate speech. This is no freak and I am convinced that any ordinary person can do the same by observing the rules of the game. Cleft-palate speech is not imitated accurately by relaxing the palate alone; alterations in the movements of the tongue have to be made in order to make the mimicry perfect. Again, it is generally recognized that after repair of the palate, some in whom an apparently perfectly anatomical result has been attained, show gross defects in speech; others with apparently poor anatomical results speak very well indeed.

The apparatus described below was devised for the purpose of estimating the relation between the competence of the pharyngeal valve and cleft-palate speech. It consists of two sets of Wolff's bottles such as are used for promoting expansion of the lungs after operation for empyema thoracis. A small collapsible gas bag is connected by a wide bore T-piece to the entry tubes of each set. [Diagram shown.]

The apparatus is used by inserting one set of tubes into the nostrils and the other into the mouth, care being taken to get an air-tight joint between the nostrils and the tube. The bags are completely emptied of air by hand pressure and by rolling them up, a burette clip then being placed on each entry tube. The subject of the test is instructed to blow through the mouth tube and is expressly warned not to let any escape through the nose. At the commencement of the act of blowing, both burette clips are released and immediately on cessation both are again caused to compress their respective tubes. The bags are now emptied of their air by pressure, and the amounts of water driven through the Wolff's bottles are the measurements respectively of the amounts of air passing by the mouth and the nose. A normal person can fill the mouth bag without any air being forced into that connected with the nose. A cleft-palate speaker passes air into both bags. This simple test then proves that a normal speaker possesses a competent palatal valve, whereas in the cleft-palate speaker this valve is incompetent. Repeated trials have shown that the more incompetent the valve, the worse is the speech. This simple fact would hardly seem to require any proof, but a further principle of greater importance can be adduced. If, in a test with this apparatus, equal quantities of air are delivered by mouth and nose, it means that within reasonable limits of experimental error, the cross section area of escape through the pharyngeal valve is equal to the cross-section area of the tube which is in the mouth. As the cross-section area of the mouth tube can always be ascertained, and can be varied at will, this gives a ready means of deducing the size of the deficiency in the naso-pharyngeal valve. Actual tests have shown this deficiency to be as small as 48 sq. mm. in good speakers after operation. Supposing the naso-pharynx to be only 24 mm. in its transverse diameter (a gross under-estimation of its size), this means that the palate fails to meet the posterior pharyngeal wall by an average of 2 mm. The small size of the defect necessary to maintain noticeable cleft-palate speech is certainly very striking.[1] Any operation designed to repair the palate should aim primarily at the formation of a functioning pharyngeal valve and must take into account the extraordinary exactitude required to attain a perfect result. It is this failure to appreciate that the pharyngeal valve is such an instrument of precision, which is partly responsible for the deplorable functional results of operations as done at present, and it may be laid down that perfect function can never be attained unless a perfect anatomical and physiological valve be first formed. The converse of this is not equally true; good speech must always be acquired by unlearning the old methods and learning the new, a want which is best supplied by the training of a good elocutionist.

Anatomy of the Defect.

There are two main deficiencies in cleft palate:—

(*a*) Defects in the soft parts.
(*b*) Defects in the skull bones.

(*a*) *The defect in the soft palate* is one which requires no great amplification. It is enough to say that there is a certain amount of loss of soft tissues; this loss makes itself very evident

[1] (Let me here add a word of warning. No accurate estimation of the palatal deficiency can possibly be attained by looking into the mouth; even the normal palate in the closed position does not appear to touch the posterior pharyngeal wall, its free lower edge and the uvula both standing off a considerable distance. So also after repair of a cleft palate the eye cannot possibly prove a reliable guide as to the efficiency of the closure of the pharyngeal valve.)

during and after operations for repair. It is impossible to compute the amount of this loss in terms of linear dimensions, but examination of cases after repair by the Langenbeck method shows the following deficiencies :—

(1) The soft palate does not reach the posterior pharyngeal wall.
(2) The repaired palate is usually a more or less rigid structure which, instead of taking a curve downwards and backwards, lies almost horizontally.

(*b*) *The Defect in the Skull Bones.*—One striking fact is the apparent increase in the antero-posterior and transverse diameters of the naso-pharynx in cleft palate. In order to ascertain how far this was due to bony defects, the following measurements were taken in normal and cleft-palate skulls, and are tabulated below :—

(1) Glabella to occipital point. } These measurements were taken for estimating
(2) Greatest transverse diameter. } the average size of the skull.
(3) Middle of the anterior margin of the foramen magnum to the hamular process.
(4) Distance between the two hamular processes, measured from the point where the tendon of the tensor veli palati grooves the bone.

Normal skulls	A	B	C
Glabella to occipital point ...	190	195·5	180
Transverse diameter	147·5	141·5	141
F.M. to hamulus	41·5	43	36
Transverse hamulus	36	32·5	30

Cleft palate skulls	Nubian, R.C.S. 210	Teratolog., R.C.S. 201	D.U.C.M.
Glabella to occipital point ...	175	187	187
Transverse diameter	128	150	145
F.M. to hamulus	43	45	45·5
Transverse hamulus	41	42	43·5

It is unfortunate that so few cleft-palate skulls are available. Measurements from two were supplied by Mr. George, from the Museum of the Royal College of Surgeons, by kind permission of Sir Arthur Keith ; the third skull is in the Durham University College of Medicine, the gift of Professor Howden. It will be observed that in all cases of cleft palate, the measurements between the foramen magnum and the hamulus, and between the two hamular processes are increased. The increase in the antero-posterior measurement is less than in the transverse. The bony deformity is undoubtedly of small dimensions, but reference to the tests of competence mentioned above shows that cleft-palate speech will result from a defect that is as small as an average of two millimetres. It is more than possible that the bony separation is the direct result of the loss of muscular pull due to separation of the halves of the soft palate. It is certain that the width of the pharynx is increased, a fact which can be easily verified by examination of almost any case of cleft palate. The reason why the operations of the present day so often fail is apparent. I know of no treatment which takes into account both the shortening of the soft palate and the deformity which is known to exist in the bones of the skull with its consequent increase in the transverse and antero-posterior diameters of the pharynx. This alteration in the conformation of the bones is of more than academic interest, and has a great bearing on the failure of restoration of speech after operation. Firstly, increased distance between the hamular processes means increase in the naso-pharynx with a consequently larger gap to be obliterated during speech. Secondly, the tendons of the tensors of the palate pass around the hamular processes, each of which is already widely separated from its fellow on the opposite side, and is also at an increased distance from the posterior pharyngeal wall. Any repair, therefore, of the soft palate in which these muscles are left intact will inevitably result in the palate not only remaining like a bow-string, under great tension, and therefore rigid, but also in its being held forward and prevented from reaching the posterior pharyngeal wall.

Is There a Solution to the Problem?

The ideal to be aimed at in the treatment of cleft palate is twofold, viz.: The prevention of regurgitation of food into the nose, and the production of perfect, not fairly good, speech. I submit that any operation which does not comply with the principle above enunciated will fail in one or both of these respects. Any valve which is to be restored must be a functional valve; therefore the use of inert material incorporated in the mobile structures is to be

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avoided. The problem resolves itself into the reduction of existing deformities in the bone and soft tissues and closure of the naso-pharynx by a physiological valve. The following operation was devised for this purpose:—

The first stage has for its object the decrease in the width of the pharynx (side to side) and the formation of a muscular buttress on the posterior pharyngeal wall to meet, and close with, the back of the soft palate.

The patient being lightly anæsthetized, and the head thrown well back, the pharynx is tickled in order to cause the superior constrictor to contract and form its transverse ridge. The position of this is carefully noted, and the anæsthetic then pushed to a deeper stage. A tenotome is now used to make a transverse incision a little over an inch long, about the level of the ridge previously noted. The incision passes through the superior constrictor muscle into the loose cellular tissue, and the point of a curved aneurysm needle is used to separate the muscle through this line of cleavage. Separation is effected both up and down and laterally as far as can be conveniently done. The wound is held into a diamond shape by means of small hooks. Catgut sutures are now inserted in order to close the wound in a vertical direction as in the operation of pyloroplasty, except, of course, that the reverse effect is desired.

Modifications of this operation can be suggested, but many attempts to obtain the desired result (in the cadaver) led one to adopt the method above described in the living body. The result of the operation is twofold; the pharynx is narrowed from side to side and a well-marked ridge, not normally present on the posterior wall of the resting pharynx, is produced. One had thought to obtain the same result by the injection of paraffin or the use of fat grafts, but finally rejected this as not being in accordance with the principles laid down. Possible dangers in this operation might be expected from retropharyngeal cellulitis or hæmatoma, or from anomalous vessels encountered behind the superior constrictor.

The second stage is the repair of the palate and can either be done at the same sitting or deferred to a later date. (For my part I found the first stage a sufficiently exacting procedure for one sitting.) The important step in the second stage is either division of the tensors of the palate or detachment of the hamular processes. The operation is to be combined either with Langenbeck's or Gillies' method of repair. Of the relative values of these two procedures in this connexion I venture no opinion.

No claim is made to the discovery of the solution of the problem of the treatment of cleft palate. It is hoped, however, that those cases in which the result is now described as good, may in future be described as perfect, and that the remaining host of poor speech results may obtain some benefit from the procedures above described. Personal experience of one recent case is insufficient evidence upon which to lay claims, but it can be stated that the operation is feasible and certainly produces immediate improvement in speech.

Dr. TRUMAN W. BROPHY (Chicago).[1]

"Lack of time and absence from my office forbid any proper assembling of my figures, but this much I did the other day: I wired to the following surgeons, asking them to give me figures which could later be classified according to ages, showing the number of Brophy bone operations they had made, with results as to mortality and necrosis, and their replies are below. These are men who have a correct understanding of the technique of the operation and who apply it daily in their large practices. I can add to this list very considerably with my own personal operations and those of many other surgeons of wide experience.

	Cases	Necrosis	Mortality
Professor A. L. Frew, Baylor University, Dallas, Texas	200	1	0
Professor S. L. Silverman, Emory University, Atlanta, Georgia	87	0	0
Professor W. L. Shearer, University of Nebraska, Omaha, Nebraska	500	0	0
Professor C. J. Lyons, University of Michigan, Ann Arbor, Michigan	900	0	0·005
Mr. G. B. Speer, Los Angeles, California ...	19	0	0

[1] Communication by letter owing to inability to attend the Meeting.

The foregoing is a report on 1,706 cases, made by reputable surgeons who, with one exception, are on the faculty of a university, showing one case of necrosis, which Professor Frew gave as 'slight necrosis,' and no deaths from this operation except in Professor Lyons' report, in which he says, 'less than one-half of one per cent' in his 900 cases. These are the operations made, not by the surgeon who originated the technique, but by men who studied it thoroughly and follow it carefully.

The essential points in maxillo-palatal surgery are the following:—

(1) Union of the separated bones of the palate and alveolar processess, with special attention to preservation and correct posing of the premaxillæ, to be obtained before closure of accompanying cleft lip.

(2) Bringing the malposed palatal plates of the maxillary bones downward by always passing the wire sutures *above* the alveolar processes instead of through them, thus approximating the edges of the cleft for one-third or one-half its length. Passing the sutures as directed, high up above the alveolar processes and immediately beneath the floor of the orbit, will obviate any disturbance of teeth. Any such disturbance of teeth has no doubt resulted from a lack of familiarity with the correct technique.

(3) Prevention of spreading of the tuberosities, by controlling and immobilizing them at the time of the anterior closure, thus laying a dependable foundation for the construction of a normal soft palate by the avoidance of the cause of tension and shortness when united, and by the provision in its stead of a long, flexible, soft palate. Once the bones are placed and fixed in normal position, the muscles will also be in normal position. Such a palate is essential for correct speech."

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